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1945

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VESICATION AND SOME VESICANTS

BY

M. W. GOLDBLATT

Manchester

Introduction

In recent years the incidence of dermatitis has increased enormously both in the chemical industries and in the ever widening field of other industries in which chemical compounds, particularly organic, have been applied. In the chemical industry the sequence of appearances known as dermatitis is so common that it is possible to see a great number of cases of much interest without forming a picture in the mind of more than the outward manifestations.

If one were to describe in the shortest terms the outstanding characteristic of so-called industrial dermatitis, it is almost certain that after sufficient experience one would wish to emphasize its 'non-specificity.' No one seeing these cases frequently can fail to be impressed by the similarity of the appearances and the diversity of the exciting agents. Whilst chronic affections, including such widely different conditions as acneiform eruptions and epitheliomata, are evoked by chemical agents, the reactions to an enormous number of widely differing chemical compounds are so similar that the term 'non-specific' can justifiably be applied to them. Such eruptions are, in general, of the acute variety and receive appellations such as papular, papulovesicular, vesicular, bullous, and so on. The signs of these acute eruptions are similar to those of eczematous eruptions, erythema, macule or raised macule, papules, small or medium-sized vesicles, bullae, oedema, etc. The symptoms are irritation, itching, prickly and burning sensation, tightness. Naturally, these signs and symptoms are mixed in different proportions in different cases depending on the individual constitution, on the site of the eruption, on the degrees of contamination, on the rapidity of treatment, on the superadded injury from scratching and home treatment.

Features of this type of dermatitis are the tendencies to set up a polyvalent sensitization, to recur and to appear in parts remote from the site of the original contamination even after removal from contact. The pathology includes spongiosis, parakeratosis, acanthosis and other features associated with eczema. The changes occurring in these cases may be divided into—

(1) vascular changes,
(2) movements of fluid (oedema, vesication),
(3) cellular changes (including sensitization), and
(4) repair processes.

In this paper we are concerned with the mechanism of vesication and with illustrations of it in the action of certain industrial compounds of which we have experience.

Mechanism of Vesication and Blistering

The phenomena of vesication are the common result of so many different agents that one is impelled to imagine that there must be an underlying mechanism common to all. Vesication can be produced by heat, cold and ultra-violet light; by friction; by such well-known vesicants as cantharides, chloroform, mustard oil and so on.

The action of chemical irritants, with which we are mainly concerned, is usually described as causing vascular spasm later followed by excessive dilation of the underlying vessels of the skin. This dilation brings about a great transudation of fluid. The lymphatics threading the epithelium also dilate and the prickle cells are so devitalized that rupture of the intercellular bridges follows and a vesicle forms in the epidermis (MacLeod and Muende, 1940). The accompaniments of the formation of a vesicle are progressive weakening of the prickle cells and cellular degeneration. This intra-epidermal vesiculation precedes the formation of a bulla as the exudation of fluid continues, provided the prickle bridges yield. The vascular dilation in the papillary layer is possibly in part brought about by a reflex motivated by the irritant on the sensory endings in the corium.

The location of the skin attacked by the irritant determines whether the bulla will develop within the epidermis or beneath it. This follows from the variations in mechanical resistance of the prickle layer in different parts of the integument. In the epidermis of the palms or soles the prickle layer is tough whereas on the face, flexor surfaces of the arms and legs and on the abdomen it is relatively weak. Thus, as fluid transudes from the papillary vessels it will tend to push up the whole thickness of the epidermis of the palm or sole giving rise to a bulla of which the roof is the whole epidermis and the floor the upper region of the corium. In other regions, e.g. face, dorsum of the hand, etc., the bulla will develop in the epidermis with the more or less devitalized stratum corneum and stratum granulosum as the roof and layers of the stratum malpighi as the floor. Thus are formed the thick-walled and

thin-walled bullae of which examples will be given below. It may be helpful to recall that the thicknesses of the epidermis of the palm and soles in the adult may be about 0·8 mm. and 1·4 mm. respectively, whereas for the other parts mentioned above it varies between 0·07 mm. and 0·12 mm. (Maximow and Bloom, 1934).

The theory of Lewis (1927) is well known. He held that there is no basal distinction in the ultimate mechanism whether the eruption was that of herpes, pemphigus, bullous or any other form of vesication. The blister, he held, like the wheal is a manifest sign of increased vascular permeability. It requires no theory to tell us that when a pocket of fluid develops there must be a source of that fluid and that in the case of a blister that source must be the vascular supply of the skin. Lewis held that there arose a state of increased permeability which was part of his well-known 'triple response.' This response—in some sense a protective response—consists of three parts, dilation of the minute vessels of the skin, increased permeability of the vessels and then a dilation of the arterioles. All these effects are attributed to the release of H-substance which in its action resembles histamine. The effects of H-substance are limited in severity by the advent of a refractoriness in the vessels to H-substance (the refractoriness is really tested against histamine). Thus, in the case of a wheal there appears to occur a rapid outflow of fluid from the over-permeable vessels and a rapid decline in the vascular permeability due to the onset of refractoriness. The blister, however, arises from a slow transudation and a much more gradual establishment of refractoriness so that the passage of fluid can continue until a pressure is developed sufficient to force apart the epidermal layers. It will be seen later in our description of cases that the time relations of blistering agents do in fact always point to a slow onset of the effect.

We have to ask ourselves how, if we accept Lewis's theory, the vesication comes about. First of all, the substance must penetrate the epidermis and reach living cells. In order to injure the cells there must ensue either an attack on the cells from outside or a penetration within the cells or both. To penetrate to the cell-interior the toxic agent must be water-soluble or it must undergo some metabolic change which leads to water-soluble bodies. Most vesicants are not water-soluble but in varying degree oil and fat soluble. In consequence of this property, vesicants can more or less readily penetrate to the living cells whereas water-soluble compounds cannot normally do so. This penetration is probably to be regarded as occurring through the openings of the sweat and sebaceous ducts, where conditions favourable to absorption are very different from those obtaining on the cornified layer of normal skin. But it should be kept in mind that water-soluble gases can pass into the skin, e.g. HCl gas. Powerful water-soluble agents attack the surface of the skin, e.g. caustics. A water-soluble compound, even in high concentrations, if pricked through the skin is rapidly diluted and diffused, whereas an oil-soluble compound will remain in concentrated form and more or less in situ.

Thus, a tentative picture of the vesicant action of an oil-soluble compound is the following:

(1) Applied to the skin, it has the possibility of penetrating to the living cell layers.

(2) Having penetrated, the concentration remains high for considerable periods.

(3) If the compound is stable, it will exert its effects by attacking the cell surfaces.

(4) If the compound is unstable in aqueous media, it may liberate water-soluble bodies which may penetrate the living cells and exert the toxic effect (cf. old theory of action of mustard gas which attributed effect to liberation of HCl).

(5) By injuring the living cells, H-substance of Lewis is liberated and this brings about the triple response.

(6) The localization of the irritant, the protracted action and the consequent maintained liberation of H-substance give the required conditions for a gradual increase in vascular permeability and hence the much slower onset of refractoriness. Fluid, therefore, continues to exude until the epithelial layers are forced apart with formation of a vesicle or blister. (It is significant to point out that Lewis was unable to produce blisters with histamine. However, this may be due to inability to simulate a sufficiently gradual introduction of the material.)

This view of blistering is of great physiological interest but it is still, as far as we know, only a theory. Lewis himself was quite aware that there might be other factors involved and was careful not to promulgate his H-substance as a single agent. Histamine salts, like other electrolytes, are not absorbed through the skin and do not blister. A recent case of my own is worth recording as showing that histamine can certainly penetrate to some extent, but this probably occurred through the openings of ducts.

Histamine Case

A young girl laboratory assistant, aged 16 years, dark-reddish hair with fair skin, gave a personal history of sensitivity to strawberries, sticking plaster, eggs (?). She had experienced 'heat spots' on occasion. No sensitivity, however, to shell-fish. She is engaged in a laboratory where virus work is carried out but had never previously had a rash on her skin whilst working in the laboratories.

One morning, in the ordinary course of her work, she weighed out 800 mg. of histamine HCl and dissolved it in 10 ml. distilled water. She spilled some of the solid compound on the bench and wiped the bench with her handkerchief. She then went to the refrigerator and on her way back to the laboratory she used her handkerchief to wipe her right eye. She experienced no symptoms.

Ten minutes later a friend drew her attention to her right eye which now appeared inflamed and swollen with considerable lachrymation. There was no pain.

On examination, I found marked oedema of the right upper lid, extreme injection of the right conjunctiva and marked erythema round the orbit. Pupils were normal. Left eyelids, etc., completely unaffected. Having concluded that this was a histamine reaction, no treatment was given and the girl was kept under observation.

Five hours later the oedema of the upper lid was diminished but still present, the injection of the conjunctiva had completely disappeared, but there was slight residual erythema on the cheek and around the orbit. Next day all signs had disappeared.

Comment

There can be no doubt that this was due to histamine, the whole triple reaction being manifest. The girl showed no greater reaction to histamine applied by scratch method than did a normal control girl. The dilutions used were (1–50) $\times \frac{1}{10^{-6}}$.

The intense reaction was, in my view, due to an enormous dose rubbed into the skin with the absorption of probably a small amount.

Demonstration of histamine or histamine-like substances in the skin is not a simple matter and should not be accepted without very sceptical scrutiny. Haas (1941) reports increases in the histamine content of the skin produced by various agents, among them such vesicants as mustard oil, cantharides, capsicum, croton oil, and turpentine oil. Moss (1942) went so far as to administer histaminase to persons susceptible to Rhus toxicodendron and found that only one out of eight cases treated (45 units per os for 25 days) subsequently developed the dermatitis when camping in an area thick with the irritant plant. We are, however, not over sanguine that such prophylaxis would prevent the development of the severe blistering of powerful vesicants.

Some Comments on Vesication

As already pointed out, there is, in general, considerable delay between the contamination of the skin and the advent of pain, heat and blistering. The rapidity of onset of these signs and symptoms seems reasonably to be attributable to the speed of absorption of the vesicant and the attainment of a concentration in the skin sufficient to irritate the endings of the sensory nerves and to injure the living epidermal cells. The well-known difference in speed of absorption of Lewisite and mustard gas sufficiently explains the more rapid onset of signs and symptoms of contamination with the former. The rapidity and completeness of healing after vesication depend upon the degree to which severe damage to cellular and vascular elements in the skin occurs. Scar formation depends upon the depth to which the vesicant penetrates and the degree of injury to cellular and vascular elements. If the base of a vesicle or bulla is formed by the corium or if the papillae are exposed, scarring will follow, but this will not result if a floor of epidermal cells remains. With severe injury to subjacent layers, there is a greater susceptibility to infection and its sequelae. There may be severe blistering as judged by size and extent without subsequent scarring. Contact of skin with a vesicant when either the skin or the vesicant is at an elevated temperature permits more rapid penetration and more severe vesication. The thickness of the wall of a blister depends upon the location, e.g. blisters on the dorsum of the hand are thin-walled, those on the palms or soles are thick-walled.

The fluid in a blister is plasma and the process of coagulation can be often observed. In cases where an inflammatory reaction occurs blister fluid will contain leucocytes and other blood cells; in some cases erythrocytes may be found.

Retention of even a small dose of a vesicant in gloves worn to protect against irritants will often produce extensive vesication, whereas had no gloves been worn an equal contamination would probably have been removed by wiping or washing. The condition of the skin inside a rubber glove is favourable to maximal effects from a vesicant or generally dermatitic agent.

Vesicants in industry are only rarely successfully inactivated in time to prevent the reaction of the skin. In the rare cases where pain is rapidly experienced (e.g. dichlor chlor-vinyl arsine) or where the contamination is observed immediately, it is possible to inhibit penetration by inactivating agents.

The process of blister formation may be regarded as in some sense protective, serving to dilute the vesicant and to prevent its further penetration. A competition may be envisaged between the penetrating vesicant inwards and the out-pouring of fluid from the over-permeable vessels. In spite of vesication from highly toxic substances, systemic symptoms do not in general appear. The possibility of systemic poisoning from arsenical vesicants has been emphasized in various publications (e.g. Medical Manual of Chemical Warfare, H.M.S.O., 1940) and theoretically this is reasonable. In the several cases of vesication due to arsenical vesicants which we have observed (*vide infra*) there have not been systemic symptoms but, nevertheless, the recommendation as to evacuation of arsenical bullae should be adhered to.

Treatment of Vesication

The appearance of severe vesication may give rise to considerable anxiety both in the patient and in the industrial doctor. The tendency is to interfere too much. If the blisters are left intact, the time comes when the enclosed fluid begins to seep through minute perforations in the epidermis. So long as the increased permeability of subjacent vessels or the inflammatory process continues fluid will be released. If the area involved is very great as in severe and extensive burns or scalds, this continued loss of fluid has its well-known consequences. In the vesication usually met with in industry the areas involved are not extensive and hence systemic effects are not likely to occur from the mere loss of fluid.

The procedure followed in dealing with the blistered skin is that applied in the case of burns and the simpler the method the better and the quicker the results. The first thing to realize is that, whatever the cause of the blistering, the tissue affected is considerably or completely devitalized and hence is more liable to infection than is normal tissue. Secondly, the contents of the blister offer a rich nidus for the growth of organisms. If, as was usual in our many cases, the blistered skin is

seen before spontaneous oozing of fluid begins, the first thing to do is to take measures to sterilize the skin surface. The difficulty of achieving this is amply dealt with by Colebrook (1944). In industrial conditions the maintenance of bacteriological cleanliness in the strict sense is probably not attainable. On the other hand, maintenance of freedom from pyogenic organisms is not only attainable but is essential for efficient industrial practice. The criterion of all good industrial medical practice is the rapidity with which workers are returned to work in a fully restored condition. In the case of vesication the absence in general of maintained subjective symptoms permits of the uncomplicated application of sound methods for assuring a bacteriologically healthy condition. The examination of the affected parts can be made without any handling, but if handling is considered necessary the nurse or medical officer should scrub up with as much care as would be considered necessary for an open wound.

Since, substantially, the vesicant action is of the nature of a second degree burn, it is relevant to quote Logie (1944): 'The second degree burn heals well with many and varied applications provided they do not offend surgical principles . . . '

The work of Colebrook has shown that the 'resident flora' of the skin (staphylococci and diphtheroid) cannot be eradicated by the ordinary use of soap and water or by the common chemical agents used for this purpose (alcohol, iodine, mercury salts, hypochlorites, etc.) since none of them appears to be able to reach the organisms in the depths of the sebaceous glands and hair follicles.

Our practice is to clean the skin with soap and water or surgical spirit to remove superficial contaminations, being careful not to break the blisters if thin-walled. If the skin is contaminated with oil, grease, chemical compounds, suitable solvents may be required to remove them as, for example, trichlorethylene or sulphonated vegetable oils for oils and greases, benzol or acetone for certain organic intermediates. Where thick-walled blisters are present there is less likelihood of breaking them in the early stages. The skin is now dried with sterile gauze. The area is now treated with 0·1 per cent. acriflavine or 1 per cent. cetavlon (trimethylcetyl ammonium bromide). All recent evidence is in favour of the latter preparation as a skin disinfectant, the combination of its detergent and antibacterial properties being happy. Not much advantage is to be expected from actually washing the skin with an aqueous solution of acriflavine, but washing with cetavlon gives a valuable soap action and by leaving the solution to dry on the skin organisms are prevented from growing in the fluid later discharged from the blister. Cetavlon is a protein precipitant and hence it might have been expected that in the presence of serum there would be considerable loss of anti-bacterial properties, but Colebrook considers that significant anti-bacterial properties remain in spite of this fact.

The skin being cleansed, the next step depends upon the nature of the blister. If there are several large thin-walled blisters it is our practice to evacuate them with a hypodermic syringe to collect the blister fluid for analysis if the presence of the vesicant or its derivatives is to be determined; otherwise the blister is incised at several points to permit free drainage. The wall of the blister is allowed to collapse upon the subjacent tissue and is not cut away. The whole blistered area is now covered with sulphonamide tulle and bandaged firmly. Small blisters are not interfered with. The bandage is left in situ for at least 2 days before examination and change of dressing.

In the case of thick-walled blisters we prefer not to drain since the likelihood of introducing organisms from the skin surface appears to us greater. After washing and, as far as may be, sterilizing the skin, a sulphonamide tulle dressing and a firm bandage are applied and left in situ for 2 days.

The healing of the thin-walled blister with replacement by healthy epithelium is relatively rapid and clean. The thick-walled multiple blisters, on the palm of the hand for example, are followed in a few days by massive cracking of thick skin and pain and discomfort on extension of fingers. There is considerable brown discoloration with wrinkling of the devitalized skin. The regenerating skin seen in the fissures of the cracked and separating skin is pink and tender. At this stage we allow spontaneous separation to proceed and apply sterile olive oil or a bland ointment (e.g. boroseptic ointment, P. D. & Co.) with comfortable bandage.

During the stage of separation of thick blistered skin, especially on the palms, stretching of the skin frequently leads to slight bleeding at the raised edges of the cracks. When this occurs over extensive areas it is desirable to swab them with cetavlon before applying the bland dressing.

The final healing of an area of thick-walled blisters, in general, requires weeks of treatment and care. In the cases we have had to deal with, chemical agents were the cause and the patients engaged in chemical work. Although we have in some milder cases permitted continuance of work with added precaution, the better rule is always to remove the case from work until final healing has occurred. In the organic chemical industry, particularly, the establishment of a polyvalent sensitivity from even slight contamination of the healing skin must be guarded against and, hence, the patient must be kept away from work. After the healing process is satisfactorily completed it must still be remembered that great precaution is necessary after return to work with organic compounds.

Clinical Investigations

The following cases give a description of vesication and present features of interest to medical personnel engaged in the chemical industry, not only because they are intrinsically significant as illustrating the effects of a variety of compounds but also because they serve to emphasize various ways in which skin injuries arise and the need to practice constant vigilance if such are to be avoided.

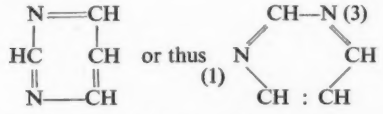
The vesicant compounds, the action of which is dealt with here, arise in certain organic manufacture, in agriculture, in intermediate stages of studies on war gases and comprise the following:

A. Derivatives of pyrimidine.
B. Derivatives of alkyl sulphides.
C. Derivatives of arsine.
D. Organic mercury compounds.
E. Organic isothiocyanates.

A. Derivatives of Pyrimidine

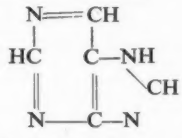
Pyrimidine is a stable, well-crystallized compound, soluble in water giving a neutral solution. Melting point of pyrimidine is 22° C. and boiling point 124° C. It forms salts with acids.

The structure of pyrimidine is represented by the formula



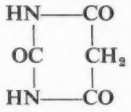
the nitrogen atoms being numbered as shown.

The pyrimidine ring will be familiar as being present in uric acid and purine derivatives, the parent compound, purine, being

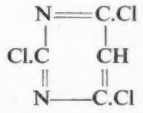


a cyclic diureide derived from a hydroxy dibasic acid and urea.

The identity of the carbon-nitrogen ring of pyrimidine with that of barbiturates will also be recognized: barbituric acid is



The first vesicant to be dealt with here is the trichloro derivative of pyrimidine—2.4.6-*trichloro-pyrimidine*,



This compound distils as a colourless liquid which solidifies at room temperature, contains 58·04 per cent. Cl and is volatile, lachrymatory and lung-irritant.

Case 1. M. P., girl laboratory assistant, aged 17 years, fair skin and hair.

1*st contact*. Engaged in distilling this compound, she spilled one or two drops on her hands. She immediately felt a burning sensation between the fingers and the affected areas developed some reddening which, however, was not very marked.

Vesication was found the next day on the 2nd, 3rd and 4th fingers of the left hand. Having been dressed at home with zinc ointment, the blistered areas were rendered septic.

Treated on 3rd day after accident in the factory medical department by cleansing, acriflavine and a dressing of sulphanilamide tulle; observation and redressing daily led to healing and recovery in 24 days from date of contact.

2*nd contact*. Fifty-one days after the complete healing of blisters, this girl was again engaged in distilling the same compound. No contact with the liquid occurred but a small amount of vapour escaped which did not produce irritation of eyes, nose, or throat. In the evening of the same day, i.e. *some* 9 *hours after the distillation*, she noticed an eruption on the hands.

When examined next day, there were a small vesicular eruption on the hands, a large bulla on one thumb and a group of vesicles on the forehead where some crusting had also occurred.

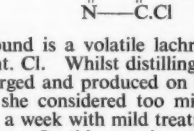
Treatment and healing were straightforward and uneventful.

The case was of interest not only because of the vesicant action of the compound but because the concentration of the vapour at the second contact was too low to affect chemists working in the same region. It would seem tentatively reasonable to consider that the young woman had developed a hypersensitivity for she certainly had been exposed to at least equal concentration on previous occasions without any reaction. Further, the concentration had been too low to lead to lachrymatory effects. The distribution of the lesions due to the vapour was also much more extensive than a small escape of vapour would normally produce.

Cases of an acquired hypersensitivity after an initial attack from a direct irritant are very common in the organic chemical industry and serve to emphasize the need to take special precautions afterwards.

Case 2. M. D., lady chemist, aged 24 years, light brunette, sensitive to elastoplast.

1*st contact*. This chemist had worked on pyrimidines in general and not specially with the chloro-compounds. Three months prior to the second contact (*vide infra*) she had prepared 2S-methyl-4-chloro-6-methyl pyrimidine,



This compound is a volatile lachrymator and contains 20·3 per cent. Cl. Whilst distilling the compound some vapour emerged and produced on the face a mild eruption which she considered too mild to report; it disappeared in a week with mild treatment.

2*nd contact*. On this occasion, three months after the first eruption, she performed an ether extraction of 2.4.6.trichloro-pyrimidine by shaking in a large flask (5 litres). She wore rubber gloves. The precise manner of contamination during this clumsy operation is not clear but within one hour from its completion she noticed a slight soreness on the palm of the right hand but continued working. Some four hours later pain and reddening had become marked on the palm and on the dorsa of some fingers. For almost two days she applied calamine lotion without relief until there was found on examination to be an extensive area of erythema and vesication on the hypothenar area, wrist, ulnar region of the palm and the dorsa of all the fingers of the right hand; there was now also small areas of vesication on three fingers of the left hand.

Treatment on the lines described continued for 11 days after which an extensive sharply demarcated area of thick palmar skin became detached with the subjacent tissue red and inflamed. No irritation of any kind was felt at this stage. The condition on the left hand was now quiescent.

Subsequent course was uneventful and there was substantial recovery in about a month from the onset.

From this case it is clear that the thio-chloro-methyl

compound vapour is an irritant, that rubber gloves did not adequately protect against the trichloro-compound, that there was a very long delay in the onset of vesication (almost 2 days) preceded by early pain and erythema. In the early stages the condition could well have been mistaken for an acute dermatitis; the pain, however, was a clear indication that deeper structures than the epidermis had been reached and that vesication was to be expected.

Case 3. G. M., laboratory assistant, aged 19 years, remarkably unreactive to known powerful dermatitic agents. Engaged in the preparation of 2.4.6.trichloro-pyrimidine, the product being extracted with ether, dried and distilled. His laboratory girl was stated to have developed blistering on the fingers after washing up apparatus used in this process.

1st contact. Some 3 to 4 weeks prior to the 2nd contact he observed the development of blisters on his fingers after handling material given to him by another assistant. This was followed by another attack some 10 days later, but the process of healing was satisfactory and he recommenced work.

2nd contact. Restarting other work with added precaution he soon developed irritant red patches on the knuckles and fingers.

On examination the appearance was that of erythematous, macular patches and no oedema, papules or vesicles were seen; there were still remains of the blistering vesicle on the web of the index and middle fingers. In the following days the eruption changed to papulo-vesicles with great itching.

Treatment was mainly with Lassar's paste and after a week recovery was sufficient to warrant return to work with instruction on special precautions required.

The interest in this case lay in the fact that the initial blistering was followed by a reaction of the kind common in the organic industry and brought about by many compounds. As already stated this boy was formerly remarkably resistant to known irritants and hence we conclude that a measure of polyvalent hypersensitivity was established as a result of the vesicant action of the trichloro-pyrimidine.

To summarize, the following can be stated:

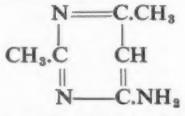
(1) Trichloro-pyrimidine is a powerful vesicant.

(2) The vesication is followed after healing by the establishment of multiple sensitivities even in persons normally very resistant.

(3) 2S - methyl - 4 - chloro - 6 - methyl pyrimidine vapour is a skin irritant, its action being reasonably attributable not only to its being a chloro-pyrimidine but also to its properties as a dialkyl sulphide.

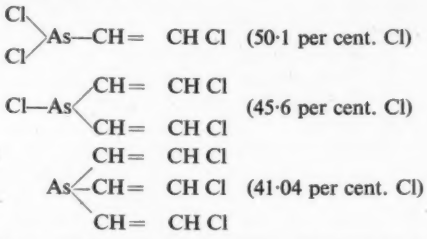
The interest in a series like this attributable without any dubiety to a single compound is to discover what radicle or group is responsible.

Such derivatives of pyrimidine as 2.6.dimethyl-4-amino pyrimidine are strong bases. The basicity here is due to the presence of the NH_2 group,



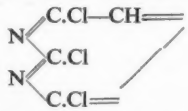
and the attack on the skin is similar to that seen with such a compound as hexamethylene diamine $NH_2—(CH_2)_6—NH_2$ and is due to the alkaline properties.

But in the case of 2.4.6.trichlor compound, the action on the skin is not due to causticity. As in the case of mustard gas or Lewisite, it is the chemical structure which is responsible. In mustard we have the structure $Cl—CH_2—CH_2—S—CH_2—CH_2—Cl$. In Lewisite we have

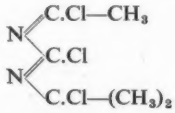


In this arsenical series the intensity of action on the skin increases with chlorination. The elements As, N, P are in the same group of the Periodic Table and hence we should expect similar properties of the chlor-vinyl derivatives.

In trichloro-pyrimidine we have the group

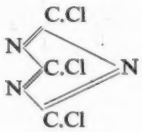


Looked at in this way and having regard to similar solubilities in organic solvents, it is not surprising that both di- and trichloro-pyrimidine are powerful vesicants. The severity is not so great in the case of the cyclical pyrimidine compound. A compound of the type

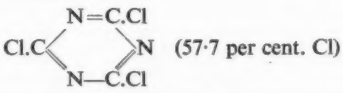


might be expected to have very severe effects.

It is relevant here to examine a compound of very great toxicity (M.L.D. for mice—5 mg./kg.), cyanuric chloride



usually written



The only difference between this compound and 2.4.6.trichloro-pyrimidine is that the nitrogen atom in the 5 position is replaced in the latter by CH. Cyanuric chloride is also a lachrymator and an irritant to the eyes and nose. We have only had one case in which the vapour of this compound emitted during weighing it out was sufficient to produce a rash on the neck and behind the ears with irritation in a laboratory assistant (fair haired and with marked acne). There seems little doubt that closer contact with this compound would produce marked vesication.

FIG. 1.—*Case 4.* Vesication produced by n-butyl-b-chlorethyl sulphide. Left wrist affected. Right wrist unaffected.

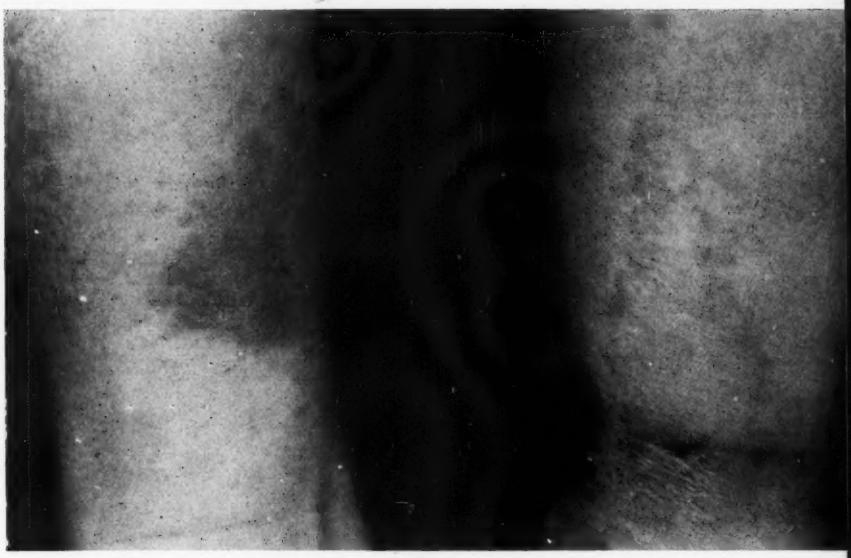


FIG. 2.— *Case 4.* Left wrist—extension from previously blistered area after exposure to sunlight. Right wrist—oedematous, erythematous area after exposure to sunlight; area protected by wrist-strap pale. 9 days after Fig. 1.

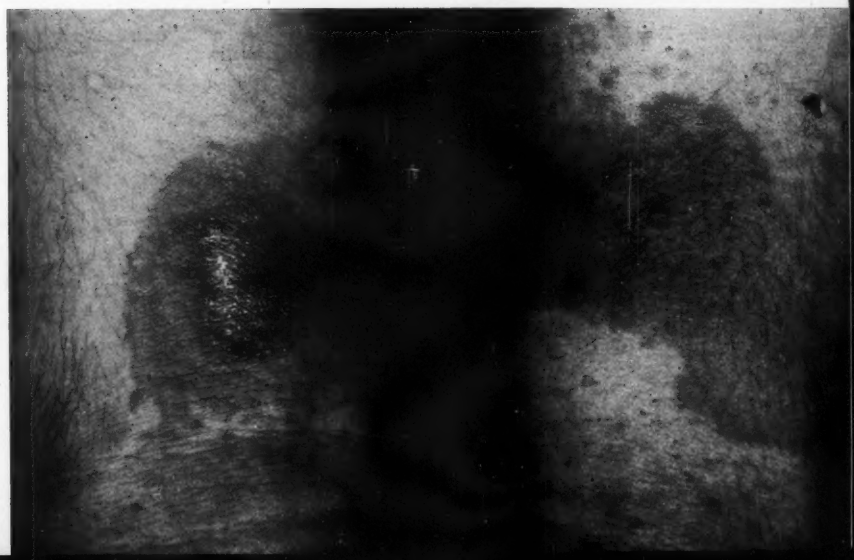


FIG. 3.—*Case 4.* Left wrist and forearm—red with surrounding brown discoloration. Right wrist and forearm—eruption after exposure to sunlight. 28 days after Fig. 2.

FIG. 4.—*Case 6.* Erythema and vesication produced by phenyl arsine dichloride.

FIG. 5.—*Case 6.* Vesication on nose and forehead produced by phenyl arsine dichloride (vapour).

FIG. 6 (below).—*Case 6.* Point of original contamination with phenyl arsine dichloride showing puckered control area surrounded by multiple vesicles.

FIG. 7.—*Case 7.* Vesication produced by mixture of aromatic chloro-arsines.

FIG. 8.—*Case 7.* Increase of vesication after removal from exposure—associated with oedema of both forearms.

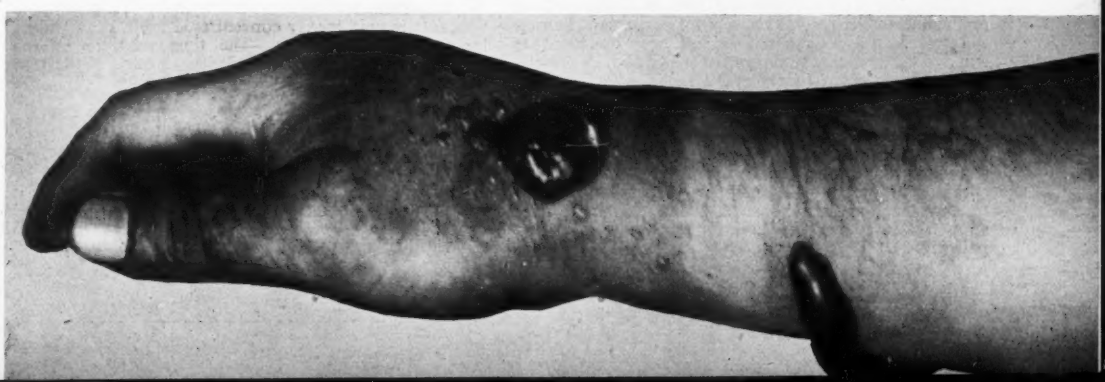
FIG. 9.—*Case 15.* Vesication produced by phenyl mercuric acetate.

FIG. 10 (below).—*Case 16.* Vesication produced by ethyl mercuric phosphate.

FIG. 11.—*Case 17.* Intense vesication produced by methyl thioisocyanate—later followed by complete denuding of palmar skin of both hands.

FIG. 12.—*Case 18.* Unusual case of vesication after intimate and protracted contract with solution of benzoin in benzene.

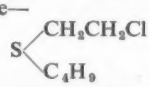
FIG. 13 (below).—*Case 18.* Tense plasma-filled bullae—area protected by wrist-strap pale and unaffected.



B. Derivatives of Alkyl Sulphides

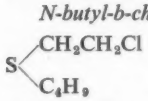
The importance of chlorine as an element which in the organic series can bring about an enormous increase in irritant properties is well known. The example of the chlorinated hydrocarbons will come to mind, e.g. chlornaphthalenes and chlorinated diphenyl. The increase in irritation which follows the introduction of 2 atoms of chlorine into the β positions in diethyl sulphide is well known. But it is always to be remembered that $(C_2H_5)_2S$ is itself an irritant, as indeed are all compounds of type

where R, R^1=alkyl radicles, inorganic elements or aromatic radicles. We recently had opportunity to observe closely the irritant effects of a compound resembling mustard gas in structure, viz. n-butyl-b-chlorethyl sulphide—



This compound contains a little more than half the weight of chlorine per molecule than does mustard gas (23·3 to 44·7). In spite of this, as we shall see, the vesicant action is extremely powerful.

N-butyl-b-chlorethyl sulphide



(23·3 per cent. Cl)

Liquid, sp.gr. 1·01, rather pleasant odour, b.p. about 92° C. at 30 mm. Hg.

Case 4. A chemist, H., aged 22 years, was engaged in the preparation of this compound and certain oxidized derivatives. Having prepared the compound, he vacuum distilled 133 g. of it, placed it in a flask for ½ hour and then transferred it to another reaction flask where it was heated with glacial acetic acid and hydrogen peroxide. A chemist nearby complained of his eyes at this time but H. was not disturbed by the vapour.

That evening at home he noticed a small blister on a red patch on the flexor surface of his *left wrist*, where he normally wears a wrist strap and watch.

At 2 a.m. that night he found 2 large blisters in the region corresponding exactly to the position of the wrist strap (fig. 1). The *pain* of the lesion awakened him. The blisters were tense with yellow fluid and were surrounded by reddened skin.

After examination and photograph, blister fluid was removed by hypodermic needle. The epithelium was allowed to remain in situ and affected area dressed with sulphanilamide tulle. Progress was satisfactory and uneventful. The area was examined and dressed daily. Patient was taken off work.

Nine days later the affected area was denuded of epithelium and surrounded by a brown-red area. During the preceding 2–3 days, patient had been much in the sun and became very sunburnt. *He had also, on instruction, treated the wrist strap with bleach; after drying he had put it on the other wrist.*

Condition now was:

(1) Left forearm—extending from the previously blistered area there was now a linear, red, slightly raised serpiginious area which was not irritating.

(2) Right forearm—flexor surface—red, hard, oedematous rectangular area above a pale area where wrist strap was in contact, i.e. area not exposed to sun was pale (fig. 2).

Treatment and progress were uninterrupted and uneventful.

Twenty-eight days later the patient stated that he had a sun-bath in his garden and, after about 3 hours exposure to hot sun without his shirt on, he noticed a red rash consisting of separate spots on his *right forearm*, flexor surface, extending from the middle of the forearm to above the elbow; there was some itching. The spots became confluent that night and on the following day there appeared a *slightly raised pink eruption* with some papule formation but no vesiculation. No other eruption elsewhere except slightly on the wrist. Originally affected area of the left wrist (*vide supra*) was now pink with brown discoloration surrounding it (fig. 3).

This case shows several of the characteristic features of the sequelae of contamination with compounds of the chlor-alkyl sulphide class as follows:

(1) N-butyl-b-chlor-ethyl sulphide is a powerful vesicant showing time relations of effects similar to those of mustard gas.

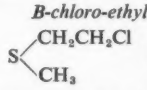
(2) It can slowly penetrate leather.

(3) A small amount of irritant material remaining after treatment of the leather strap with bleach set up an inflammatory reaction on normal skin.

(4) After substantial healing of the blistered area, there remained a marked photosensitivity.

Contamination of the skin by penetration through a wrist strap is unusual, but among workers it not infrequently happens through fabric or leather gloves or through defective rubber gloves. The lesson is obvious.

B-chloro-ethyl-methyl sulphide



(32·1 per cent. Cl)

This compound by virtue of its lower molecular weight contains a much higher proportion of chlorine than the n-butyl compound.

Case 5. H. F., undergraduate chemist, aged 19½ years, was engaged in the preparation of vinyl sulphones. His chemist was handling b-chloro-ethyl-methyl sulphide wearing rubber gloves.

At between 10 and 11 a.m. one morning, H. F. inadvertently touched his chemist's rubber glove and a drop of the above compound remained on the extensor surface of the right thumb. The drop of liquid was washed from the skin some little time later.

About 5 hours later H. F. observed a small patch of redness on the skin but was not discomforted. His chemist advised him at this stage to put bleach on it; this he did and allowed the bleach to dry on the skin.

Some 12 hours after the contamination 2 closely lying small blisters appeared on an erythematous base. The blisters coalesced about 14 hours after the contamination and there was some pain on stretching the skin.

Four days of treatment on lines outlined found the blistered area clean and led to uninterrupted recovery.

In the crowded conditions of war-time, events of this kind have not been infrequent, chemists and others being affected by materials with which they were not concerned.

It is not easy from this case to state whether the compound involved was more or less violent a vesicant than the n-butyl compound. The effect of the contamination was very local and bore out the view that the oil-soluble vesicants remain locally concentrated until, presumably, inactivation takes place. The time relations of the blistering process were again similar to those of mustard gas.

C. Derivatives of Arsine

As, fortunately, there has not been any gas warfare in the recent war, opportunity to see the effects of various types of war vesicants has not been afforded. Nevertheless, much research went on from 1940 on this kind of substance.

The arsenical smokes received a good deal of attention, in particular the aromatic arsenical cyanides. The study of intermediate stages in the manufacture of these and later the production of these intermediates in bulk came within our sphere of control. It may be mentioned in passing that in the course of this investigation (2–3 years) many tons of As_2O_3 passed through the shed specially allocated for this work. Regular medical examination of the workers was instituted and, considering the rather crude manufacturing methods used, it was a source of no little surprise that no cases were discovered. After a few months of manufacture we sketched a method of handling (or rather non-handling) of the arsenic which was quickly installed and perhaps contributed to the complete absence of any case of arsenical poisoning throughout the 3 years work. The final products, $C_6H_5.As : Cl_2$ and $C_6H_5.As : O$, were handled in the plant in such a way that no cases of skin injury were met with among the workers. But on the research side cases were found among the chemists and we propose here to give an outline of the phenomena observed.

Phenyl arsine dichloride. $C_6H_5AsCl_2$ (M.A.).

Case 6. R. S. N., chemist, aged 26 years, fair type; as a child he had attacks of urticaria and was subject every year to hay-fever.

Whilst working on the preparation of M.A. a small liquid contamination occurred on the right forearm; this led in a short time to erythema and a few small vesicles appeared here and on a finger of the left hand. Continuing at work small serous vesicles appeared on the right temple.

In order to observe the development of these lesions no treatment was given and he continued at work with, however, added precautions. On the fifth day from the first contamination a crop of vesicles on a red base was present on the forehead and nose and erythema had spread to the flexure of the right arm with multiple vesicles on the forearm (figs. 4 and 5). The point of original contamination on the right forearm now showed a puckered central area surrounded by a red area sown with minute vesicles (fig. 6). Local irritation was slight but greatly aggravated in bed. On the following day there was a spread of vesication round the site of the original contamination and of erythema to the back of the neck. Treatment was now commenced with Lassar's paste and Veganin, the patient being put to bed as he had developed a cold.

During the next 12 days there was, in spite of removal from all possible contact, spread of vesication preceded by development of erythema until on the 19th day all areas had practically completely recovered, a fine desquamation of most areas continuing for some days. In the later stages dressings of oil and calamine were used.

Reviewing this case we may note the rapid vesication following the original contamination and the great spread that took place due, doubtless, to continuance at work in low concentrations of vapour. Sensitivity to such concentrations was certainly increased by the establishment of the original lesion. It is to be noted also that new crops of vesicles occurred after removal from exposure which suggests a measure of generalized sensitization.

About a month after the first vesication above, the patient was working with diphenyl chlor arsine, $(C_6H_5)_2AsCl$ (D.A.), and a splash of liquid occurred on to his left forearm. It was *immediately* treated with sodium hypochlorite but, nevertheless, in some 10–15 minutes irritation began and a flare of erythema appeared from elbow to wrist on the ulnar aspect. Itching and erythema continued for 4 days when vesicles began to develop on the reddened area. On the 5th day the erythema had been replaced by groups of small vesicles located mainly round the hair follicles. There was no irritation at this stage and recovery took place after about 12 days. Considering that inactivation by hypochlorite was immediate we must consider that the speed of penetration was very great and that D.A., left in situ, would produce severe results.

Phenyl arsine dichloride and diphenyl arsine chloride.

Case 7. D. S. P., chemist, aged 24 years, dark hair and skin, skin rather dry. This man was engaged with a variety of arsenical compounds and at the time of the events described below was handling the following 4 compounds: $C_6H_5AsCl_2$, $C_6H_5.AsO$, $(C_6H_5)_2As\,Cl$, $(C_6H_5)_3As$.

In spite of having had his hands, wrists and lower forearm protected with rubber gloves, contamination by one or more of these compounds occurred.

It will be simple to visualize the sequence by tabulating the appearances on succeeding days.

Day	*Appearances*
1	Experiment performed but no ill-effects noted.
2	He observed slight redness and itchiness on right wrist.
3	Right wrist began to swell but there were no vesicles.
4	Confluent masses of vesicles appeared on right forearm and wrist and vesicles on red base on dorsum of right hand. One medium-sized vesicle surrounded a white raised rim on a reddened base appeared. The *left* forearm and wrist were similarly but less severely affected. Some of the vesicles had already broken down and small cracks were left in the skin. On the face there was a circumoral somewhat itching papular eruption. *Patient was removed from all exposure.*
5	Erythematous area extended with vesication on right wrist; half-inch blister with large number of minute and small vesicles around it. Similar development to medium-sized vesicles on dorsum of right hand. On left hand and wrist erythema with slight development to vesiculation. Face and chin, no further development.
6	Oedema of right forearm with increase in vesiculation. On left forearm large blister just above dorsal wrist, surrounded by raised pale area in turn surrounded by ring of small vesicles (fig. 7). *Right leg* now showed patch of erythema halfway down from the knee on tibial surface.
8	Marked further development of vesication on dorsa of both hands and on flexor wrists. Oedema of both forearms (fig. 8). *Supratrochlear glands enlarged on both sides.* No great discomfort. Patch on leg not further developed.
9	Vesication still marked by improving. Some extension to flexures of both arms. Supratrochlear gland on left arm larger. Oedema still present.
10	Great improvement. Glands still palpable but diminished in size.
18	Dorsa of hands and wrists show red, roughened, cracking, inflamed skin but there are no subjective symptoms.
22	Substantially recovered. Treatment on lines given. For the face, always a difficult problem in industry, the treatment was liq. alum. acetat. after cleansing; no covering was used.

The precise manner in which the original contamination came about was not discovered. There is a good deal to be said in favour of doing work of this kind without gloves: arrangements for plunging the hands or arms straight into a bath of inactivating solution immediately a drop of any material being handled alights on the skin being more likely to preclude the effects of contamination unperceived if gloves are worn.

We cannot judge as to the amount of material which contaminated the skin, but from the long delay before the first signs or symptoms appeared we conclude that the subsequent severe effects were due to the establishment of hypersensitivity to the low concentrations of vapour which are almost inevitable in work of this kind.

The development of blistering after removal from exposure, remote from the point of original contamination (probably right wrist) must indicate a wide dissemination of the irritant or a derivative of it or conceivably the release of some locally formed allergen. The patch on the right leg suggests that, had work been allowed to continue, there would have arisen a very widely distributed affection.

The enlargement of the supratrochlear glands is, in our experience, an unusual accompaniment of even very severe dermatitis or vesication, provided, as in this case, there is no sepsis.

We regard this case as one of vesication from a mixture of arsenical vesicants with a secondary sensitization.

Case 8. F. R. C., chemist, aged 29 years, dark skin and hair, no history of sensitivities. Engaged in the analysis of the 'mixed oil'—$C_6H_5AsCl_2$+C_6H_5.As.O.

During some 3 weeks of work of this kind this man experienced no more than slight irritation and reddening of the face from which there was no further development.

One morning, without any warning or knowledge of contamination, he observed a single blister, 3–4 mm. in diameter, on a slightly reddened base. This was situated 2½ inches above the wrist on the left flexor forearm. There was no irritation. We opened the blister, removed the skin and dressed with 1/3000 $KMnO_4$. In spite of no spread or complication the lesion did not heal for over a month at which time there was still an erythematous patch with a central scab.

We mention this case to illustrate how long a time even an apparently trivial lesion due to organic arsenicals can take to heal.

Reviewing these and other cases of arsenical vesicants, we find it difficult to conclude as to which of them is the most potent. The dichloro compound contains 31·8 per cent. Cl and the monochloro 13·4 per cent. Cl, but this is not a sufficient criterion as to vesicant properties. As we shall see, other chlorinated organo-metallic compounds possess vesicant properties and the latter certainly do not depend only on the chlorine present for they are maintained with radicles other than chlorine, e.g. acetate, phosphate, nitrate. The nature of the organic radicle is of great importance. Although more study is required, it is our view that aliphatic organo-metallic compounds are more toxic than aromatic compounds. Such compounds are, however, very sensitive to water, e.g. Lewisite, methyl and ethyl arsine dichloride—$(CH_3)AsCl_2$, C_2H_5.$AsCl_2$. The chlorine atom attached to the arsenic is very sensitive to water and is readily hydrolysed by it. For this reason the cyano derivative was a principal subject of study since it (a solid) is stable to and insoluble in water. This compound, one of the nose-irritant 'smokes,' has, however, diminished vesicant effects in comparison with the chlor-compounds.

It seems justified to say that the chlorine atom increases the vesicant action, and that the oily compounds are more active than the solid compounds. But it must not be supposed that solid compounds cannot be powerful vesicants (*vide infra* organic mercury compounds). Oil solubility is an important accessory property for vesicant action as opposed to water solubility.

D. Organic Mercurial Compounds

Considerable medical interest attaches to organic mercurial compounds. Hunter *et al.* (1940) have described in detail clinical and pathological effects which were produced in workers engaged in the preparation of methyl mercuric nitrate (CH_3—Hg—NO_3) and in monkeys exposed to the vapour. They also made short reference to the effects which compounds of this type could produce on the skin.

The development of organic mercury compounds for the protection of seed against attack by fungi constitutes an extremely interesting field in the scientific methods of plant protection and has contributed in vast degree to the increase of yield of a considerable number of different crops.

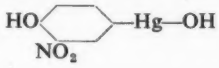
A large number of preparations containing organic mercury compounds has been marketed in various countries under a more or less confusing variety of names. It is often difficult to determine what the constituents of these products are. The object of all such preparations is to make possible a destruction or inhibition of growth of fungi which, if allowed to act on the seed, produce diseases in the plant leading to serious diminutions in the yield and, by offering them a satisfactory nidus for growth and multiplication, maintain the infectivity of the soil. This goal must be attained without injuring the seed, tuber or bulb or the process of germination. There are many techniques of attack on and defence against fungi and insects in the soil. Among these are the chemical treatment of the soil itself or of the seed, tuber or bulb. The particular chemical compounds used naturally depends upon their ascertained or claimed efficacy against particular insects or fungi known to attack crops in which the consumer is interested. They may be used in steeps, sprays, powders or dusts and it is both in the manufacture of the insecticide or fungicide and in its diluted applied form that medical hazard arises.

In greater or lesser measure all mercurial compounds are hazardous. Having regard, however, to the very large quantities used in agricultural and horticultural practice and the relatively small numbers of cases of toxic effects met with, it may be concluded that if the directions of the makers and distributors are followed no great anxiety need be felt in the utilization of most organic mercurials.

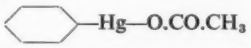
Most organic mercurials of the general formula R—Hg—R′ where R may be a hydrocarbon (aromatic or aliphatic), a chlor-phenol, a cresyl or tolyl residue, a nitrophenol, and R′ an organic or inorganic anion (e.g. —CN, —SO_4Na, —OH, —H_2PO_4, —$OCOCH_3$, Cl, etc.) are vesicants and it is of the effects of some of these that record is made in what follows.

Used in the form of dusts diluted with an inert powder the mercury contents of different preparations vary considerably but modifications are

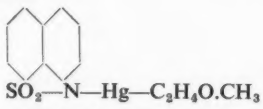
frequently made and marketed under the same name. Thus, Uspulun (Tillantin R) of probable formula



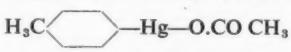
and Ceresan



both German preparations, were at one time marketed in dilutions containing 3·4 per cent. and 1·5 per cent. Hg, respectively, but at a later date a new Ceresan appeared which was an entirely different compound, viz. methoxy-ethyl mercuri-naphtha-sultam



which, in the diluted form for use, contained 1·2 per cent. Hg. Agrosan, a British product, contains



probably a mixture of isomers and is marketed containing 1·5 per cent. Hg, whereas a Ceresan made in Germany for the U.S.A. market contained 2·0 per cent. Hg. The matter is complicated further by the fact that proprietary preparations contain more than one mercurial.

Attempts have been made to relate mercury content and molecular structure with fungicidal activity. Some evidence that increase in the molecular weight of R decreases fungicidal properties has been obtained but other factors than this enter and complicate the problem of practical application. Similarly, the content of mercury, important as it must surely be, is not readily to be regarded as determining fungicidal activity. The actual build-up of the molecule has great significance. As to the vesicant and dermatitic effects of these compounds, it is unfortunately the case that the users of this kind of compound are not as conscious of the need to apply the measures of protection seriously recommended by the manufacturers and always appear to have heard of these for the first time when workers are adversely affected. The fact is that of the preparations which ultimately reach the market in quantity, none will affect more than a minute fraction of persons using them if proper precautions are applied. From time to time, one sees reports of agricultural workers being burnt by seed dressed with mercurial compounds. A report appeared that a man had lost his life through 'having his hand burnt by mercurial salts while sowing a field of wheat' (*Farmer and Studbreeder*, 1943). No details are given about this case, but we should venture to doubt whether the 'mercurial salts' were indeed the cause of death.

The author had an opportunity to attend an inquest on a farm worker when it was alleged that his handling of seed dressed with mercurial compounds had contributed to his death. The cause of death in this case was septicaemia following an injury contracted during an attack on a rat in a farm shed.

Poisoning of farm animals and poultry is sometimes reported as resulting from the ingestion of grain dressed with mercurial fungicides. Thus, Edwards (1942) reports the case of a bay gelding of 16 hands, aged 9 years, which the owner stated had eaten a quantity of oats dressed with a mercurial, the quantity being 'not more than a quarter of a bucketful.' Edwards reports that the condition of the horse was that of advanced mercury poisoning —'loose bowels,' anorexia, depression, dejected appearance, unsteady gait, increase in rate of pulse and of respiration, temperature 104° F. Gangrene of the lungs was evident from the foetid sweetish breath coupled with a bloody, muco-purulent nasal discharge. The horse was dead next morning. Edwards notes the very small quantity of treated oats which suffices to poison so large an animal. Edwards also refers to fowls being poisoned from eating grain treated with mercurials. Such events in human subjects have not, as far as we know, ever been reported.

Apart, however, from the effects of these compounds as systemic poisons, there is need to consider their action on the skin since their widespread application brings cases to the notice of industrial and other medical men which may present difficulties both in diagnosis and treatment. The cases we describe occurred both in the use of such compounds already diluted with inert powders in agricultural work and in the mixing of them with diluents in works conditions.

Case 9. *Case of handling dressed seed.* F., 31 years old. Intelligent, healthy young farmer. Fair hair and skin.

In October, 1941, he was sowing seed in the course of which he had carried on his back sacks of seed dressed with a mercurial containing tolyl mercuric acetate. Twelve months prior to this he had observed a mild skin eruption during a similar operation, but this had passed off without much trouble. The present rash, however, was much more extensive. It consisted of 'spots' on his chest, back, groins and arms but not on his face and hands. The rash was not itchy.

His doctor diagnosed scabies and prescribed hot soda baths, scrubbing of skin and Proscabin. This resulted in the most maddening irritation and itching which became progressively worse. A consultant saw the condition and diagnosed it as eczema-dermatitis and treatment with Proscabin was stopped. The legs now began to swell and the condition became so bad that morphia was given and on some occasions the patient had to be tied down to the bed. A consulting dermatologist now saw the case and took him into hospital where he remained for 13 to 14 weeks. Every kind of blood and other tests were performed. X-ray therapy, at first not very successful, later apparently led to great improvement (3 or 4 exposures at intervals of 7–10 days) with diminution of irritation and gain in sleep. A patch test with the mercurial seed dressing (details not known) gave an erythematous reaction. (A control test on another man led to blister formation. A medical officer at the hospital also applied the preparation to his own skin and reported intense irritation.) During the course of hospital treatment there was extensive exfoliation of the skin which, in the patient's own words, came off 'in buckets.'

In September, 1942, patient left the hospital much improved and returned to light work, avoiding all contact with mercurials. Some 3 weeks afterwards he observed a reappearance of the condition and now, in addition to the former areas, the hands and face were also affected but in less degree. On examination at this stage by the writer it was found that in the axillae, umbilical region, belt line, anal cleft, buttocks, the skin was purple-red in colour, cracked, peeling and in the axillary and pectoral regions intensely lichenified. The back and arms were less severely affected with many small vesicles at all stages of development. The dorsa of the hands were covered with minute vesicles. The legs and ankles were covered with an angry eruption of small vesicles at all stages.

Diagnosis and Comment

The condition when first seen by the writer was that of a severe chronic generalized inflammation of the skin, showing all the stages associated with a sensitization dermatosis. Some areas were obviously in the stage of acute exacerbation. The course of events point strongly to the original eruption having been due to the chemical irritant, but that the mistaken diagnosis of scabies and the violent treatment which followed it led to a condition of sensitization dermatosis. The latter continued for the long period described and recurrent exacerbations were to be expected. The fact that recurrence occurred without further exposure to the original irritant indicates that the sensitivity was now polyvalent. Whilst the vagaries of human skin do not permit us to rule out a polyvalent sensitivity from the original irritant, it is a fact that eruptions from organic mercurials are not characterized by the establishment of such a sensitivity if properly treated from the outset.

Case 10. *Case during dressing of seed.* M., 23 years old, fair haired, very healthy looking man.

This man was engaged in the actual dressing of seed with the mercury compound (containing tolyl mercuric acetate). He had done similar work in 1937 and 1938. In 1941, he resumed this kind of work. One day he noticed some non-itching, non-irritant *blistering* between the fingers of his right hand: 'the skin seems to just curl up and come away.' He also developed an eruption on the anterior neck; this started as a red patch and now appears as a localized group of broken-down vesicles. The condition was diagnosed by his doctor as ringworm and treated as such. The treatment made the eruption much worse and when seen by the writer he was being treated by ultra-violet irradiation.

Diagnosis and Comment

As in the former case, the doctor's diagnosis was certainly incorrect and due probably to lack of acquaintance with the effects of organic mercurials. The treatment would certainly involve the risk of establishing a sensitivity as in the former case.

Case 11. *Case during dressing of seed.* P. Not seen by writer.

This man was a soldier who had been released from the Forces to do seed-dressing work. After a week's work on seed dressing he developed *blisters* on his hands and had to leave work. The blisters were described as big ones, tense with fluid. It appears that the seed-dressing machine had become partly blocked with paper and stringy material; P. put his bare hand into the machine to clear the obstruction and it became contaminated with the seed-dressing preparation. The skin blistering followed in a few hours. The further course is not known.

Examination of the seed-dressing plant at which Cases 10 and 11 had worked showed the reddish dust of the mercurial in considerable amounts strewn about the apparatus and the floor. At one point there was continued egress of the powder during the running of the machine. In spite of the warning instructions issued to the seed dressers, personal precautions were very poor. The manager of the unit in spite of his skin already being affected, handled the preparation with the bare hands with the utmost unconcern. The bags containing the dressed seed were satisfactorily dust proof, a point of considerable importance since otherwise the mercurial dressing tends to 'bloom' out on to the outer surface of the bags.

Whereas the complaint met with among a small proportion of persons coming into contact with this mercurial compound is one of irritation of the skin, occasionally other symptoms are mentioned.

Case 12. *Case during dressing of seed. Firm H.*

Here men engaged in dressing seed with a mercurial had formerly complained only of skin irritation similar to that of cases described above. But it was reported that a recent consignment of the preparation had led to *sickness*, *diarrhoea* and *loss of appetite*, in addition to sneezing which, it was stated, was less pronounced than with previous batches of the material. This last statement is indubitable evidence of dissemination of the powder in the working place and indicates a gross neglect of the precautions emphasized by the makers.

Case 13. *Case during dressing of seed. Firm S.*

Here a man who had been in charge of seed dressing with a mercurial for several seasons without any skin affection found, subsequently, after some weeks of such work that the skin of his hands began to peel between the fingers and his eyes became irritated and painful.

Case 14. *Case during dressing of seed.* C. H., 22 years old, strong, healthy man, brunet type. No history suggestive of skin allergy.

He was transferred from field work to assist in seed dressing and dressing operations in a granary. The machine used was of a standard type and elementary precautions were taken to prevent dissemination of mercurial dust in the working place. He was engaged in filling sacks with dressed seed from the seed-dressing machine. Whilst the filling of the sack proceeds (4 bushels) he steps outside the immediate area of the sack, which is surrounded with fabric screens. When it is judged that the required weight of seed has entered, the man steps forward and shuts off the flow of grain, shakes the bag down, puts the bag on the weighing machine and adjusts the weight with a scoop. The bag is now tied and removed to a convenient spot.

After 9 hours of this work on the first day of his work in seed dressing he had dealt with 46 bags of grain. Having washed with cold water and soap, he went home without any complaint of skin irritation or discomfort. Next morning on waking his hands were blistered. He was sent to hospital where he was diagnosed as 'suffering from injury in the nature of a superficial skin burn associated with using a chemical seed dressing.' He was healed as an out-patient. The lesions were washed with ether and treated with picric acid. Progress was good.

The only subjective symptom complained of in the early stages was a sensation of heat some 12 hours after ceasing work. At no time was there complaint of pain and the patient slept through the night without discomfort whilst the lesion was developing.

When seen by the writer the superficial blistered skin had been removed from the fingers of both hands and the subjacent tissue was pink, healthily epithelializing, clean and dry. No other signs or symptoms were or had been present and recovery from the skin condition was uneventful. Recovery took about a month. The patient had been warned by the doctor never to undertake seed dressing again.

It is important to note that neither this man nor any of the others engaged in seed dressing wore protective garments of any kind. None of the other men had, however, suffered in any way from their contact with the seed-dressing material. The affected man had hot and perspiring hands to which, he stated, the powder tended to stick.

The cases above described were due to dressing or handling dressed seed with a preparation containing totyl mercuric acetate. These were all men meeting the material during its agricultural application. Men engaged in the actual manufacture of organic mercurials are so guarded against contact by the nature of the plant that it is uncommon to come across a case of vesication. Opportunity has, however, presented itself to observe the effects of two other organic mercurials, phenyl mercury acetate and ethyl mercury phosphate.

Case 15. *Phenyl mercuric acetate—*

$$C_6H_5—Hg—O.CO\,CH_3.$$

G. S., aged 40 years. Process worker.

This man was engaged in grinding phenyl mercuric acetate in an experimental plant where the rigid precautions necessary in dealing with such compounds had not yet been introduced in the design of the plant itself. The worker wore tightly fitting rubber gloves but appears at some stage of his work to have got some of the powder into the gloves without noticing it. No immediate symptoms were experienced, but some hours later on washing his hands he noticed redness of the skin and experienced a burning sensation. Next day there were large bullae on the dorsa of both hands and on the medial and lateral surfaces of the fingers (see fig. 9).

As the case was seen with the extensive blisters intact, the opportunity was taken to draw off some of the blister fluid with a hypodermic needle The fluid was straw coloured and coagulated in the tube yielding a serum in which analysis showed a content of 12μg Hg per g. On making an incision into the raised skin, the subjacent layer could be seen as a jelly-like mass extruding into the incised area. This mass was clearly coagulating plasma from which, on slight pressure, serum could be expressed.

Treatment with sterile olive oil and bandages led to uneventful recovery in 22 days after the usual cracking and peeling of skin had taken place. Throughout the treatment there was no irritation or sepsis.

There can be no doubt that in this case the compound was in undiluted form and in extremely intimate contact with the skin. Nevertheless, no immediate symptoms were produced, vascular dilation and vesication following after a considerable delay.

The Hg demonstrated in the blister fluid is unequivocal proof of the penetration of the compound through the skin.

In a recent paper (McCord, Meck and Neal, 1941), the effects of phenyl mercuric oleate (commercial)

$$(C_6H_5—Hg—O.CO.(CH_2)_7—CH=CH—(CH_2)_7—CH_3$$

on human and animal skin are described. These authors found that 0·5 per cent. of this compound in a mixture of paraffin oil, kerosene and acetone applied to human skin produced pain in 3 hours, great discomfort in 7 hours and in 20 hours vesication with an inflammatory areola. Control with the solvents produced no vesication but kerosene led to a definite inflammatory reaction after 24 hours. Whilst all persons tested with 0·5 per cent. and a high proportion of persons tested with 0·2 per cent. reacted in this way, no subject tested reacted to 0·1 per cent. Rabbit skin seemed more susceptible than human skin, 0·1 per cent. producing an inflammatory reaction. No further details are given of the reaction of animal skin, but we presume that vesication was not induced.

Case 16. *Ethyl mercuric phosphate—*

$$C_2H_5—Hg—O—P(OH)_2$$
$$\|$$
$$O$$

This compound, like phenyl mercuric oleate, is used as a wood preserving agent. Vintinner (1940) describes the process of immersion of pine boards in a solution containing 1 part of ethyl mercuric phosphate in 6600 parts of water Men exposing their skin to the solution developed a burning sensation from 3 hours to 3 days after contact and this was followed by oedema and vesication. The vesicles were thick walled and taut and up to 1½ inches in diameter. Lesions appeared on hands, forearms and feet; in one case a vesicle appeared on the lower lip. Disability lasted for from 5 to 30 days. The author had record of 42 cases of this kind, but it appeared that some men were not susceptible.

The preparation used to make up the preserving solution consisted of 6·25 per cent. ethyl mercuric phosphate in borax and talc as diluents.

The kind of lesions produced by this mercurial is shown in a case of our own (fig. 10). The patient was a fair-haired and fair-skinned lad of 17 years. He was a laboratory assistant engaged in the preparation of a mixture of ethyl mercuric phosphate with inert diluents in a ball mill for research purposes. He wore rubber gloves with the sleeves of his overall tucked into them. He also wore a nose pad. Contamination of the hand with the mixture nevertheless occurred and led to the usual sequence of absence of initial symptoms, several hours later a sensation of heat and irritation with appearance of erythema and still later the advent of vesicles and blisters. The figure shows thick-walled vesicles on the palm and a denuded blister on a finger tip laying bare the subjacent tissue. After the blister had become established, no pain or irritation was experienced and the condition was readily healed without scar formation on the lines laid down above.

Comments on the Handling of Mercurial Fungicides

Including the references in the literature we now have evidence of the vesicant action of—

phenyl mercuric acetate,
ethyl mercuric phosphate,
phenyl mercuric oleate,
tolyl mercuric acetate.

It seems not unreasonable to anticipate that all compounds of this kind, i.e. of the type R—Hg—R′ would, in greater or less measure, have similar effects on the human skin.

From our observations on Case 15 we conclude that these compounds are demonstrably absorbed through the skin and that the degree of absorption is increased by maintained contact, particularly if trapped inside a glove.

The circumstances in which skin effects may occur have been indicated. The actual planting of dressed seed is almost universally done by machine drilling and, hence, handling is minimized. The dressing of the seed is properly done in specially adapted machines, but the possibility of contamination by and even inhalation of dust is not eliminated. Seed-dressing stations are not in general characterized by the installations required to maintain a dust-free atmosphere. It is not likely that seed-dressing firms would look favourably on the need to set up plant assuring full protection of the workers and, hence, are likely to prefer to continue with such measures as gloves, overalls and, in some degree, warnings. It must be admitted that, if we judge by the relative numbers of cases brought to notice, the problem appears to a considerable extent to be under control. If proper attention is paid to prevent dissemination of mercurial dust and the workers wear an efficient dust mask or respirator, there should be little danger of systemic absorption. The protection of the skin is a more difficult matter. Non-fatty skin creams may have a function; even

powdering the skin with inert powders before and during work may be useful. Gloves, if used, should be of stout fabric and designed to extend well up the arm. These should be washed daily and worn during all operations involving dressing seed, handling bags of dressed seed, making up the weights of bags of seed, loading, unloading, cleansing of machines and so on. Men on this kind of work should, ideally, wear overalls with the trouser legs over top-boots. There should be facilities at hand to permit of washing away contaminations of the skin as soon as they occur. Wrongly treated, the skin lesions may be followed by sensitization, but this need not occur if the cause of the condition is realized. As with most organic compounds some persons are inherently sensitive to any degree of contact.

Although we have not come across any case of systemic poisoning from these compounds in their agricultural use, it could be considered desirable to examine the urine of workers for Hg at specified intervals. Even in the manufacture of organic mercurials under the most stringent plant precautions, it is nevertheless possible to detect Hg in the urine although hitherto we have not detected clinical signs or symptoms of toxic absorption.

E. Organic Thioisocyanates

The vesicant action of allyl thioisocyanate (allyl mustard oil) is well known. The alkyl thioisocyanates are often referred to as a class as mustard oils.

The following properties are noteworthy:

$CH_2=CH—CH_2.N:C:S$, allyl thioisocyanate, liquid, pungent odour, b.p. 151° C.

$C_2H_5.N:C:S$, ethyl thioisocyanate, liquid, pungent, b.p. 134° C.

$CH_3.N:C:S$, methyl thioisocyanate, solid, m.p. 35° C., b.p. 119° C.

Organic thioisocyanates differ from the isomeric thiocyanates in that the alkyl radical is attached to the nitrogen atom thus:

R—N=C=S—thioisocyanate.

R—S—C ≡ N—thiocyanate.

The thioisocyanates have application in certain organic syntheses and are all powerful vesicants.

The following case is described first because it illustrates the characteristic action of this group of compounds and also because of the establishment of an extreme sensitivity after apparent cure.

Case 17. R. C. H., aged 18 years, student chemist. This young man was engaged in a study of methods for the preparation of thioisocyanates.

1st contact. Whilst reacting isopropylamine with carbon disulphide in caustic soda followed by decomposition with basic lead acetate for the preparation of isopropyl thioisocyanate ($(CH_3)_2CH.NCS$), he experienced some slight irritation of the skin but this soon passed off.

2nd contact. A few days later prepared methyl thioisocyanate by same reaction using methylamine instead of isopropylamine and decomposing with ethyl chloroformate instead of basic lead acetate. On this occasion there was pronounced irritation and lachrymation (due to the chloroformate). This also passed off quickly.

3rd contact. One month after the first contact was again preparing CH_3NCS and whilst washing his apparatus noticed slight lachrymation and irritation of the fingers. The latter became worse next day and there developed small, medium-sized hard vesicles on the palms and fingers; the fingers became oedematous and there was slight itching; small vesicles appeared on left flexor forearm and left neck which cleared up in a day or two. The subsequent development on the hands, however, was severe. All work was discontinued.

Days after 3rd contact	*Appearances*
3	Confluence of vesicles with production of massive blisters involving the whole thickness of the palmar skin. No pain or itching. Fingers very oedematous with appearance of fresh blisters. Some of the thin-walled blisters on the interphalangeal surfaces oozing (fig. 11).
8	Many vesicles have drained spontaneously; considerable desquamation; oedema of hands much diminished; skin, mainly of palmar surface of fingers, stippled with irregular spots.
11	Blisters are now mainly hard lumps; skin cracking in parts with slight bleeding; at finger tips one feels, on pressure, the separation of whole thickness of skin from subjacent layers; affected parts are discoloured to brown; stippling has now gone from parts where skin is now hard and ready to detach.
14	Skin of whole affected part now thick, hard, cracked and peeling. It is clear that the whole of the palmar skin will be lost. Subjacent tissue pink, clean and dry.
15	Pain on extending fingers. Hard blistered skin slowly separating.
28	Progressive improvement maintained. Skin of hands now appears almost normal but somewhat mottled.

Thus, in about a month there had been complete detachment of the entire palmar skin of both hands and re-establishment of apparent normality. Treatment had been on the lines already described and there seemed no reason to anticipate further difficulty. Patient was, however, not permitted to recommence work in organic research.

Nevertheless, some 7 days later, the patient, on entering the laboratory for some hours where work on thioisocyanates was proceeding, developed an exacerbation of the skin of the palms of both hands. The skin became itchy, hot, swollen and tight and massive blisters developed on palm and fingers. Treatment was required for 14 days.

The importance of this case lies mainly in the extreme sensitivity which remained after the initial severe vesication had completely disappeared, for the second attack occurred without any manifest contact with the original vesicant except the slight concentration of vapour in the atmosphere of the laboratory. It seems clear that even slight concentrations of vapour can penetrate the skin and evoke (directly or indirectly) increased vascular permeability, which is a necessary prelude to vesicle formation, in cases of extreme sensitivity such as that described above.

This case re-emphasizes the need for extreme caution when deciding on the return to chemical work after an attack of severe vesication. The same caution is, indeed, required after any severe attack of skin inflammation due to chemical irritants.

The nature of this caution is :

(*a*) To be assured that the skin is completely restored to, at any rate, apparent normality;

(*b*) to permit return to work at first without any exposure to chemical irritants, in order to

ascertain whether the mere act of work is supported by the skin;

(*c*) to permit later return to work with compounds other than that which brought about the original attack, in order to determine whether a polyvalent sensitivity has become established;

(*d*) to permit later return to original work first more or less as an onlooker and, if no exacerbation occurs, as a full participant with precaution.

Proceeding in such a way may, perhaps, be regarded as an attempt at desensitization and in a considerable number of cases success is attained but many cases, in spite of long delay before return to normal work, retain an untractable sensitivity and must be transferred to non-chemical work. We have seen cases maintaining hypersensitivity twenty years after the original attack by a particular compound.

It is not our purpose to discuss the question of idiosyncratic sensitivities but one further case may perhaps be of interest as showing a high degree of sensitivity to compounds which ordinarily do not act as vesicants.

Case 18. McK. Process worker. Poor physical condition.

This man had worked in an experimental chemical plant for 5 days preparing diphenylacetophenone ($C_6H_5COCH(C_6H_5)_2$. In the ordinary routine of the plant he wore gauntlet gloves but being inexperienced he turned the gauntlets down and some material from the reaction mixture entered and remained in close contact with the skin of his right hand and lower forearm.

Diphenylacetophenone is formed by the condensation of benzoin ($C_6H_5CHOH.CO.C_6H_5$) with benzene in presence of concentrated sulphuric acid which removes the elements of water.

The phenomena which followed on the worker's skin render it certain that there was no contamination with the concentrated acid and we attribute them to the solution of benzoin in benzene.

Pain was not felt at the time of the contamination but was experienced on going home at end of day's work. This was followed by development of oedema and erythema of the right hand and wrist and severe vesication. The blisters were, as usual, tense with yellow plasma and an area where a strap was worn remained pale and unaffected (figs. 12 and 13).

The course was uneventful but somewhat protracted.

In attributing the case to sensitivity to the benzene-benzoin mixture we do not ignore the fact that there was extreme intimacy of contact for several hours. We have never seen blistering from either of these compounds but were not able to perform suitable tests on the patient. Benzoin is a powerful reducing agent, reducing Fehling in the cold, a fact which may be related to the effect described.

Summary

A short description of the possible mechanism of vesication is given.

A technique for treatment of chemical vesication is described.

Certain compounds met with in the organic chemical industry which are powerful vesicants are considered. These are:

derivatives of pyrimidine,
derivatives of alkyl sulphides,
derivatives of arsine,
organic mercurial compounds,
organic thioisocyanates.

The establishment of hypersensitivity to chemical compounds after the apparent healing of a vesicated skin is a frequent event and special precaution is required in deciding on the return to work of the affected person. In certain cases photosensitivity may follow vesication.

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CONTROL OF HAZARDS IN THE LUMINOUS DIAL PAINTING INDUSTRY

BY

J. C. JONES and M. J. DAY

From the Department of Physics, Middlesex Hospital Medical School, London

The paint which is applied to the dials of instruments so that the figures and pointers shall be visible in the dark consists of a mixture of zinc sulphide with a very small proportion of radium sulphate. A proportion of seventy millionths of a gram (70 micrograms) of radium element to 1 g. of zinc sulphide is commonly used. Zinc sulphide alone will glow in the dark after it has been exposed to light, but the luminosity disappears after a few hours. To ensure a continuous luminosity the admixture of radium is necessary. The rays from the radium which are in themselves invisible cause the zinc sulphide to give off a greenish light, a phenomenon which is known as fluorescence.

The mixture of zinc sulphide and radium sulphate is made up into a paste, usually with gum arabic and applied to the figures and pointers of the dials with small applicators made of wood, metal, or plastic. The dials are left overnight in a dark room in order that any luminosity due to the previous exposure to light of the zinc sulphide should have disappeared. They are then tested against standard dials which are illuminated from behind. The operatives who apply the paint are known as luminizers or radium dial painters, and the workrooms as luminizing rooms.

During the 1914–18 war the demand for aeroplane instrument dials which were self-luminous produced a considerable increase in the number of persons employed in the industry. In the years 1918–30 several fatalities occurred in the U.S.A. among persons who had been so employed, which were thought to be due to the absorption of radium. Most of these operatives suffered from aplastic anaemia and many of them showed bone changes which culminated in osteogenic sarcoma. An account of the early work which was done in this field and of the attempts to trace the cause of these fatalities is given by Martland (1931). The clinical symptoms did not generally appear for some years after the persons had left the work. In some cases they were delayed for as many as ten years. Since that time a considerable amount of work has been done on the subject and an account of this will be found in papers by Evans (1943) and others. A comprehensive list of references is given in a review by Read (1939).

Radioactivity

Before discussing the hazards involved in this work and the physical measurements involved in their control, it is necessary to describe the general properties of radium and allied substances. Radioactive substances, of which the element radium is the best known member, spontaneously emit radiations. These radiations arise from the disintegration of the atoms of the elements and are of three types. The alpha-rays are streams of helium atoms, each with a positive electric charge shot out from the nucleus with high velocity. The beta-rays consist of electrons, ejected from the atoms with very high speeds. The gamma-rays, which accompany the production of alpha- and beta-rays, are electromagnetic radiations, similar in nature to x-rays.

All three types of ray produce ionization in any medium through which they pass; that is, they are capable of removing electrons from the atoms of the medium and thus of transforming them into positively charged particles or ions. It is probably this property which produces deleterious effects on living tissue cells. By the production of these positively charged atoms inside the cells the chemical equilibrium is disturbed; this inhibits mitosis and, if great enough, may cause the death of the cell. This property is used to measure the intensity of the radiations. Air is normally a non-conductor of electricity, but if any of these radiations pass through it they cause it to become a conductor. If two metal plates separated by a layer of air or other gas are connected to an electrical supply no current will pass between them; but if a radioactive substance be placed nearby, the air will become a conductor and current will pass. By measuring the magnitude of this electric current the intensity of the rays from the radioactive material can be determined. From this the amount of radioactive material present can be estimated. In practice the current passing between the outer walls of a cylindrical chamber and a central metallic rod is measured (fig. 1). The radioactive material may be outside the chamber, in which case the radiations must pass through the walls; or, if it is a gas, may be within the chamber. Such a vessel is called an ionization chamber.

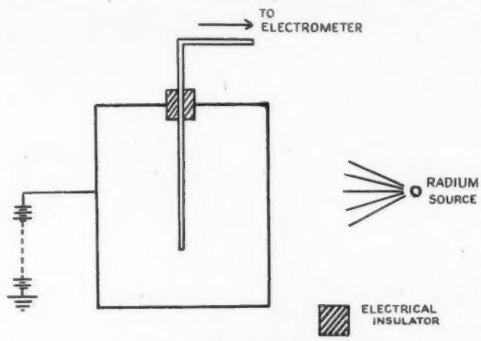

FIG. 1.—Ionization chamber.

The alpha-rays have a very low penetrating power, being stopped by a few centimetres of air and about a tenth of a millimetre of human tissue. Being comparatively massive (they are about 8000 times as heavy as an electron) they travel in straight lines and produce intense ionization along their tracks. The beta-rays are more penetrating, being able to penetrate several metres of air. The ionization along their tracks is correspondingly less dense. The gamma-rays have a much higher penetrating power and produce diffuse ionization throughout the medium.

These rays are produced by a series of disintegrations of the nuclei of the radioactive atoms. The radium atom disintegrates, emitting an alpha particle and becomes itself a radon atom. Radon is a heavy and chemically inert gas. It is itself radioactive and disintegrates, producing an alpha particle and becoming an element known as radium A. A whole series of disintegrations takes place and the final product is lead:

$$\text{radium} \rightarrow \text{radon} \rightarrow \text{radium A} \rightarrow \text{radium B} \rightarrow \rightarrow \text{radium C} \rightarrow \text{— — — — —} \rightarrow \text{lead}$$

The rate at which the atoms are disintegrating is proportional to the total number of atoms present. Thus, if the radon which is produced by a specimen of radium be collected and its radioactivity studied separately, the intensity of the rays emitted decreases with time. In 3·85 days it would fall to half its original value. The radiation from a radium tube which has been sealed for some time consists of all three radiations corresponding to the different stages in the disintegration series. Thus the gamma-rays, which are emitted from a radium needle used in therapy, are produced by the breakdown of the radium C atom.

If radium is deposited in the body, part of the radon produced remains in the tissues, and the rest is expired through the lungs. Thus the gamma-rays, produced from radium in the body, would be less than those from a sealed radium tube containing the same amount of radium, for some of the radium C, produced from the exhaled radon, is deposited outside the body. The percentage of the total radon produced which is expired, is called the emanating power of the radium deposit. This factor is important in the estimation of the quantity of radium in the body. The elements radium A, radium B and radium C are solids and are deposited when the radon atoms disintegrate. This solid deposit is usually called the 'active deposit' of radium. It is itself radioactive and capable of causing injury to the tissues if deposited in them.

The units we have used to express quantities of radium in the body and concentrations of radon gas in air are the microgram and the eman. One microgram is one millionth of a gram. The eman is defined as a concentration of one ten-thousand-millionth part of a curie per litre of air, the latter unit being the quantity of radon which produces the same intensity of gamma radiation as one gram of radium.

Hazards

The various hazards involved in the manufacture and use of radioactive substances in industry may be classified according to the radiations responsible for them.

(*a*) *Alpha-rays.*—Owing to their low penetrating power these rays can only cause damage to the tissues by the immediate contact of unscreened radioactive material. In practice this may be brought about by the absorption of radium into the body through the mouth, as dust through the lungs, by the deposition of radioactive material on the skin, and also by the breathing of air containing dangerous quantities of radon gas which is given off from the radium. The condition produced by the absorption of radioactive material into the body is known as radium poisoning.

(*b*) *Beta-rays.*—Although stopped by a few millimetres of most solid material these rays can penetrate several metres of air producing intense ionization in the region of the source. Obviously it is the hands which are subject to the greatest exposure from this source.

(*c*) *Gamma-rays.*—The harmful effects produced by gamma-rays are well known from experience in hospital radium and x-ray departments. The tolerance dose rate of one roentgen per week is generally accepted. The roentgen is the physical unit of quantity of x or gamma radiation defined by the International Congress of 1928 on the basis of the ionization produced by these rays. It gives a measure of the total energy absorbed per unit volume of tissue from the x- or gamma-rays. In comparison with other workers handling radioactive materials in this industry the risk of damage from the gamma radiation is small. In hospitals a radium officer may safely handle several hundreds of milligrams of radium for a short time. The quantity of radium on the factory bench is seldom more than 0·4 milligrams for each operator. On the other hand, in hospitals, the radium is sealed up in containers through which the alpha- and beta-rays cannot penetrate. The risk of absorption of radium into the body by hospital workers is negligible.

Methods of Protection

Safety measures necessary in industry fall into two groups. Firstly, there are precautions designed to reduce unavoidable exposure to a minimum and to improve the general conditions of work; these are laid down by the British Radium and X-ray Protection Committee (1943), and the Ministry of Labour and National Service (1942, 1943). The correct emphasis on the different aspects of protection can only be assured by quantitative measurement of the radon and radium dust in the atmosphere and on the beta- and gamma-ray intensities.

Secondly, direct examination of the operatives themselves for indications of over-exposure is necessary; this entails on the medical side routine blood examination, inspection of the hands for signs of local effects and attention to the general health of the workers, and on the physical side the wearing of beta- and gamma-ray sensitive photographic films and tests for radium in the body.

Radium Poisoning

The symptoms of radium poisoning are so severe (Browning, 1944), and the quantities of radioactive material required to produce them are so small, that it must be regarded as the most important risk run by those working with ' naked ' radium. Early work on the internal administration of radium salts for therapeutic purposes showed that not all the radium ingested is retained. At first elimination is rapid, and at the end of a few weeks, if the radium has been taken orally, only about 10 per cent. remains in the body. (About 90 per cent. of the radium eliminated appears in the faeces and 10 per cent. in the urine). The elimination rate decreases until after a year, the radium remaining may be considered as virtually fixed in the body and is to be found mainly in the skeleton with rather smaller concentrations in the lung, liver, spleen, and heart (Reid, 1939). The percentage retained is about 1–2 per cent. if the radium has been taken by mouth and 2–10 per cent. if it has been inhaled by dust.

It has been established that it is possible for 1 microgram of radium remaining in the body to cause death (Evans, 1943) though cases have been reported of persons apparently unaffected by quantities up to 10 micrograms.

From the physical point of view a microgram of radium produces ionization effects of the same order of magnitude as are produced by an x-ray dose rate of one roentgen per week, the accepted tolerance value. The effects of the alpha-rays are much more localized. Thus a very high dose rate is maintained for a very short time over very small volumes and the affected cells probably have less chance of recovery. Gray and Reid (1942) found that it required a physical dose of gamma-rays about nine times the alpha-ray dose to kill a broad bean root tip.

It is clear that in order to avoid danger to health the radium content of the body should be considerably below 1 microgram. Curtiss (1942) and Evans (1943) both suggest a maximum allowable amount of 0·1 micrograms. The British X-ray and Radium Protection Committee suggest that there should be no more radium in the body than will produce a concentration of 0·1 emans in the expired air. The relation between these recommendations is seen when it is realized that there are two methods for the detection of such small quantities of radium in the body: (*a*) direct measurement of the gamma-ray intensity produced by the radioactive substances in the body and (*b*) the measurement of radon appearing in the expired air which is proportional to the radium content of the body if the emanating power remains constant. It is clear that the first method measures the quantity of disintegration products emanating in the body and the second those given off. For a complete estimate both measurements should be made. Thus if 50 per cent. of the radon produced escaped in the expired air, a concentration of 0·1 eman in the breath would indicate a deposit of 1·6 micrograms. Variations from 90 per cent. to 10 per cent. have been found in the emanating power in individual cases. It appears to decrease with the time the radium has been in the body. It has not been found easy to measure by the gamma-ray method quantities of radium less than 0·2 micrograms. This quantity of radium would produce a concentration of radon in the expired air of 0·01 emans if the emanating power was 40 per cent. It is therefore suggested that the total quantity of radium in the body should be less than 0·2 micrograms and that the concentration of radon in the breath should not exceed 0·01 emans. (The figures refer to a time when the radium has become fixed in the body.) Considerably higher quantities are permissible if measurements can be made within a few days after the ingestion of the radium. If it is suspected that a sudden large intake of radium has taken place measurements on the expired air should be made as soon as possible; but if any ingestion takes place it is probably fairly continuous, so where a deposit is detected it is safer to assume that the radium has become fixed unless very frequent routine tests prove otherwise.

Prevention of the intake of radium through the mouth is achieved by insistence on strict adherence to working rules laid down by the Ministry of Labour and National Service. Where radium dust concentrations are appreciable, suitable face masks are useful, but it is more important to keep the radium dust to as low a value as possible. Calculation can be made of the concentration of radium dust in the air required to produce an accumulation of 0·1 micrograms of radium in the body at the end of the year. Assuming a breathing rate of 10 litres per minute, a 40-hour working week, and a 10 per cent. retention of radium (all of which err if anything on the high side), the value obtained is 0·8 micro-micrograms * of radium per litre. The radium dust concentration should therefore be kept well below 1 micro-microgram per litre.

* 1 micro-microgram = 1 million millionth of a gram.

FIG. 2.—Girls luminizing. Note protective aprons, lead glass screens in front of operatives, and local exhaust ventilation system.

FIG. 3.—Showing protection afforded by lead glass screens in front of operatives.
(By courtesy of Smith Accessories, Ltd., Cricklewood.)

Radon in the Atmosphere

A clear distinction should be made between the radon which arises from the radium in the body and is present in the expired air even when the operator is expiring normal air, and the radon which arises from the radium on the benches of the workroom and which is breathed in by the operator. It is established that the breathing of radon can produce harmful effects, but direct experimental evidence on the actual concentration required to do this is meagre. The incidence of lung carcinoma is high among workers in the Joachimsthal mines (Martland, 1931) where the radon concentration seems to average about 50 emans and occasionally rises to 200 emans. But it is by no means certain that this is the cause of the trouble, for the presence of arsenic dust in the air may have some effect. Various tolerance concentrations have been suggested for the radon in atmospheric air breathed by operatives. Mottram and Reid (1939) found that for mice a concentration of 1000 emans does not produce any appreciable effect on the growth rate for 161 days, though 5000 emans is fatal within a few weeks. Exact comparison between mice and men is uncertain, but they suggest that as it is not difficult to keep the concentration of radon below 10 emans this figure should be aimed at. The tolerance concentrations put forward by the British X-ray and Radium Protection Committee and by Evans (1943) are respectively 1 and 0·1 emans.

In practice the main justification for such a low tolerance as 1 eman lies in the comparative ease with which concentrations may be kept below this value by exhaust ventilation (Hemon and Evans, 1943; Williams and Evans, 1942). We doubt however whether as low a value as 0·1 eman can be justified as a restricting working condition.

Measurement of Radium in the Body

It is important that direct tests should be made for the presence of radium in the body in order that operatives may be put off work and possibly treated with the object of increasing the rate of elimination of radium from the body if ingestion has indeed taken place. The two available methods of measuring the quantity of radium in the body are: (*a*) direct gamma-ray measurement, and (*b*) measurement of the concentration of radon in the expired air after the operator has been breathing normal air for some time in order to ensure all the radon in the expired air does actually come from radium in the body. One microgram of radium in the body gives from 0·01 to 0·2 emans in the expired air according to the emanating power of the radium in the body.

Expired Air Tests

In spite of the variation in the emanating power, which leads to uncertainty in the interpretation of results, this method has many advantages; it is the only one which can readily be applied on a large scale. Collection of breath samples is comparatively easy whereas the gamma-ray test involves the presence of the operative at the laboratory. Accordingly, this method of detecting ingested radium was first developed and we have found it possible during the later part of the work to detect a concentration of 0·02 emans corresponding to 0·2–2·0 micrograms of radium.

Samples of expired air were collected in litre aspirators by displacement of water. The operator was asked to attempt to empty his or her lungs completely; although this does not give the concentration of radon in the normal expired air, we thought that it would be likely to produce a higher concentration than normal breathing, thereby rendering the detection of small quantities somewhat easier. Samples were collected on Monday mornings before the operators started work, after breathing normal air since Saturday. Some spuriously positive results were later traced to contamination of the atmosphere in which the samples were taken so that later samples were taken in the open air as far as possible. Control samples of atmospheric air were always taken.

Gamma-ray Tests

When testing patients, they were instructed to lie on a couch as closely as possible on the arc of the circle of a metre radius centred on the ionization chamber. Patients bathed and washed their hair after their last visit to work and, if they could not

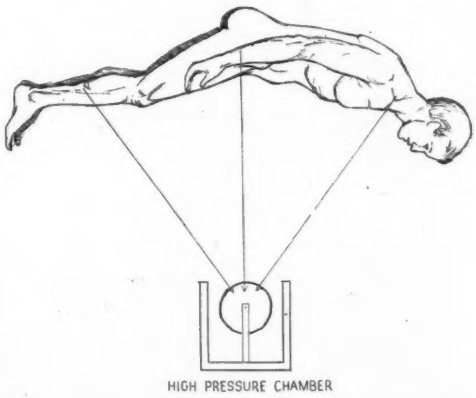

FIG. 4.—Gamma-rays from deposits of radium in the body cause current to flow in the ionization chamber.

wear clothes which had not been near the luminizing room, they changed into hospital clothing. Quantities of the order of a microgram have been found in clothing which had only been worn at work a few times and the ordinary working clothes must often contain many times this amount, since even the under-clothes have been said to glow in the dark. During the test luminous wrist watches had to be removed to a safe distance.

After measurements on the total activity with the patient one metre from the chamber, further measurements were made in different positions with the chamber close to the patient, as a test for small localized quantities of radium. One patient's

hair was found to be slightly contaminated in this way, the activity disappearing after thorough washing.

Results of Tests for Radium in the Body

The expired air from 120 operatives at 32 factories has been tested. Of these 11 showed positive results with concentrations ranging from 0·02 to 0·09 emans. Owing to the comparative insensibility of our apparatus any positive result was deemed to be significant and it was recommended that these persons be removed from work. There seemed to be some correlation between the period of employment and the positive results, all those concerned having been working with radium for more than 18 months and 2 of them for more than two years. Several of these workers were tested again, along with a batch of others, by the gamma-ray and breath methods about a year later and the results are shown in Table 1. In the one apparently positive case

TABLE 1

GAMMA RAY TESTS FOR RADIUM IN THE BODY

No.	Employment	Period of employment	Gamma-ray activity (micrograms)	Radon content of expired air (emans)	
				At time of gamma-ray test	Previous test
1	Luminizer ..	8 years	0·03±0·06	<0·02	—
2	Charge hand ..	5 ,,	0·03±0·09	<0·02	<0·03
3	Luminizer ..	4¾ ,,	0·04±0·11	<0·02	0·2
4	Luminizer ..	2 ,,	0·04±0·05	0·05	0·04
5	Luminizer ..	3 ,,	0·25±0·10	0·1	0·05
6	Luminizer ..	2 ,,	−0·09±0·13	<0·02	0·03
7	Luminizer ..	3¼ ,,	−0·04±0·06	<0·02	0·6
8	Chemist ..	12 ,,	0·10±0·07	0·06	0·05
9	Chemist ..	4 ,,	0·02±0·08	0·04	—
10	Radium technician	13 ,,	−0·12±0·08	<0·02	<0·05

The errors given are the standard deviations from the mean of several physical determinations. There is a probability of 19 to 1 that the actual values do not vary from the mean by more than twice the above limits. Negative values indicate that the instrument indicated less ionization with the patient near to it.

measurements with the high-pressure chamber close to the body seemed to indicate that the radium was concentrated in the region of the head, probably in the hair. We were unable to carry out further tests to confirm this and the breath test was inconclusive, the control sample showing that the atmosphere in which it was done was contaminated with radon. The discrepancy between the results shown in Table 1 and the earlier breath tests may possibly be explained by the contamination with luminous paint of the clothing of those tested, or by the fact that previous to the test they had been in an atmosphere outside the luminizing room where concentrations of radon were sometimes as high as inside.

Measurement of Radon and Radium Dust Concentrations in the Workrooms

Atmospheric air samples were collected from workrooms usually during the middle of the morning. Three types of workrooms were concerned in these tests. Firstly, the luminizing room where the actual dials are painted with the radium and zinc sulphide mixture. The number of the operatives in the room varied from one to thirty, but the rooms were usually large and exhaust ventilation was in use in nearly every case. Of the 37 rooms tested, 7 showed a concentration of less than 0·2 emans, 25 had between 0·2 and 1·0 emans and only 5 showed a higher concentration than this, the highest being 6 emans. It is obvious, therefore, that for these rooms there is no difficulty in reducing the concentration to less than that recommended by the British X-ray and Radium Protection Committee.

The second type was the dark room, small and often unventilated, where the luminized dials are placed over night before being tested for luminosity. Three rooms showed concentrations less than 0·2, 11 came between 0·2 and 1·0 emans and 11 from 1 to 6 emans. It should be borne in mind that the operative is only in the room for a few hours a day, but even so the concentrations seem high.

The third type was the radium laboratory and other places where large quantities of radium were used or stored. There the concentrations were much higher, only 1 being below 0·2 emans, 5 between 0·2 and 1·0 eman and no less than 7 greater than 1·0 emans, the maximum being 8·0 emans.

The concentrations of radium dust ranged from 2 micro-micrograms per litre to 10 micro-micrograms per litre. Measurements were only carried out in one factory which specialized in the production of the luminous compound, but it is very probable that the dust concentrations in luminizing rooms would be considerably smaller. The face masks used in the factory where tests were made consisted of a wad of cotton wool between layers of surgical gauze, tied over the mouth and nose by tapes (see figs. 5 and 6). They showed an efficiency of about 60 per cent. when stretched over a glass funnel at an air flow rate slower than the normal breathing rate. The efficiency of the mask must be appreciably lower in practice, since air probably leaks between the face and the mask.

In luminizing operations, the beta and gamma radiations are probably the least likely to produce serious harm.

Note on the Accidental Dispersal of Radium

During the course of this work we were concerned with two cases which showed in a remarkable manner the persistence of spilt and scattered radium. In the first of these 50 mg. of radium, in solution as bromide, were thrown from a window over a lawn. An area of about seven square yards was excavated to a depth of two feet and fresh soil put in. About three months later we examined the site with a neon tube radium finder and estimated that there were still approximately 5 mg. present.

In the second case, radium paint containing about 50 mg. of radium was scattered in a fire caused by enemy action. It was originally kept in a second-

FIG. 5.—Protective clothing worn by operators working with large quantities of dry luminous compound.

FIG. 6.—Bottle filling at a factory manufacturing luminous compound.
(By courtesy of Brandhurst Company, Reading.)

storey room. The site was cleared to ground level and left open. A good deal of the paint was salvaged. Two years after this we thoroughly examined the place and in a small area about 10 yards horizontal distance from the original store place of the radium we found indications of not less than 5 mg. of radium.

Conclusions

Measurements indicate that workers in most ordinary luminizing factories are not suffering from excessive exposure either to radon in a contaminated atmosphere or to radium dust.

In some cases the ventilation of dark rooms in these factories could be improved.

Workers with larger quantities of radium, for example in the preparation of luminous compound, are exposed to greater risks, and in these cases we regard the risk of the inhalation of radium dust as particularly important. Moreover, such workers are not protected by any special regulations.

The situation is on the whole reassuring and we consider that, if the Ministry of Labour regulations are rigidly adhered to and the necessary routine tests made, there will be no need to look on this industry as a dangerous occupation.

Summary

A short account is given of the hazards involved in radium dial painting and of the physical measurements necessary for their control. The recommended tolerance figures for the quantity of radium in the body and for the concentration of radio-active material in the atmosphere are discussed. Results of measurements made on operatives and workrooms are given.

Acknowledgments

Most of the work was carried out for the Medical Research Council at the Middlesex Hospital and was under the direction of Professor S. Russ to whom we are greatly indebted. Visits to the factories concerned were arranged by Dr. Ethel Browning of the Factory Department, Ministry of Labour. The gamma-ray tests were made possible by the kindness of Dr. L. H. Gray and the Mount Vernon Hospital, Northwood.

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METHYL IODIDE POISONING

BY

ANDERSON GARLAND and F. E. CAMPS

From Burton Road Hospital, Lincoln, and the Chelmsford and Essex Hospital

Toxicity of Methyl Derivatives

It is becoming increasingly obvious that all methyl derivatives are highly toxic substances. It has long been known that methyl alcohol is dangerous since it occupies a peculiar place among solvents on account of the temptation to use it as a beverage. The most prominent symptom has been amblyopia followed by blindness which is frequently total and permanent and caused by a primary optic atrophy, but less severe exposure may cause acute gastro-intestinal disturbance, headache, vertigo, feeling of intoxication and dilated pupils, while severe exposure may be followed in a short time by giddiness, unconsciousness, sighing respiration, depressed cardiac action, cold sweats, violent delirium, coma and death. Recently Jacobson, Russell, Grimm and Fox (1945) and Kaplan and Levreault (1945) have reported 60 cases of methyl alcohol poisoning occurring in the Pacific theatre of war where Japanese have been using the substance as a fuel. American soldiers have found drums containing remnants and used it as a drink mixed with fruit juices. Sixteen of the cases were fatal.

Røe (1943) suggested that the action of the poison could be attributed to the inhibition of oxidation processes by formic acid, which forms a complex compound with iron. The process is followed by an acidosis which is mainly due to lactic acid. Amblyopia does not occur until the acidosis is well established except in the presence of strong light. Increase of metabolism may provoke or aggravate amblyopia since it increases the acidosis. The oxidation of methyl alcohol is checked by the consumption of ethyl alcohol; and all signs of poisoning may be averted if it is drunk before or repeatedly after the consumption of methyl alcohol. Treatment consists firstly in counteracting the acidosis by the intravenous injection of isotonic sodium bicarbonate. Much fluid by mouth combats dehydration and promotes diuresis and loss of chloride may be counteracted by 5 per cent. glucose in saline. Gastric lavage should not be undertaken until the intravenous sodium bicarbonate is started. Ethyl alcohol helps to prevent recurrence of acidosis. The eyes require protection against light during the acute stage of the poisoning and if amblyopia persists.

Methyl chloride is a colourless gas with a faint ethereal odour. It is used in the dye industry and in the preparation of chloroform, and to an increasing extent it is replacing ammonia and sulphur dioxide in refrigeration. In 1927 and 1930 Baker described 96 cases of poisoning amongst employees of an American firm of refrigerator manufacturers, and Kegel, McNally and Pope (1929) gave a full account of the symptoms based on 29 cases, of which 10 were fatal. Jones (1942) gave an account of seven cases which had occurred in Great Britain among travelling refrigerator repairers. In mild cases, the symptoms include staggering gait, dizziness and headache, followed the next day by anorexia, nausea and vomiting, which are not as a rule severe enough to prevent the man from working. In more severe cases, the patient may be ill for weeks, the staggering and dizziness being accompanied by drowsiness, malaise and weakness. In about half the cases, ocular symptoms, consisting of misty vision, diplopia, and difficulty in accommodation occur after about 24 hours, and may persist for two months. Vomiting is sometimes most persistent and may last up to a week or ten days. Diarrhoea is described. In the most serious cases epileptiform convulsions occur and may lead to death. Involuntary movements include myoclonus and action tremors. Ptosis, strabismus, and diplopia are common, but optic atrophy does not occur. Slurred speech, amnesia, drowsiness by day, and delirium by night occasionally seen. The temperature, pulse and respiratory rate are raised, and in the worst cases there is oliguria and suppression of urine. Albumen and red cells occur in the urine in at least half the cases. No case has shown jaundice though fatty degeneration of the liver has been seen at necropsy. In some cases the red cell count fell as low as 3,100,000 per c.mm., and the haemoglobin to 50 per cent.; sometimes there is a leucocytosis. Sequelae such as ataxia, diplopia, misty vision, headache, drowsiness and amnesia may persist for as long as eight months. Poisoning in refrigerator factories can be prevented by adequate exhaust ventilation; but it is difficult to explain to the refrigerator repairer that an almost inodorous non-irritating gas is poisonous. He must be forbidden to stay in a room after he has heated liquid methyl chloride in the blocked evaporator of a refrigerator. Warning of a leak from a refrigerator might be given to a householder by adding a pungent detector

substance such as acrolein, but it is probable that methyl chloride will soon be replaced in refrigerators by freon (dichloro-difluoro-methane) which is equally efficient and is harmless.

Methyl bromide is an important fire extinguisher, fumigant, refrigerant, and delousing agent, and recently many cases of poisoning have been reported. (Watrous 1942, Miller 1943, Gray 1944, Holling and Clarke 1944, De Jong 1944, Heimann 1944, Clarke, Roworth and Holling 1945, Wyers 1945, Butler, Perry and Williams 1945, Michaux, Clercy and Lechevallier 1945, and Duvoir and Dérobert 1945.) The toxic effects fall into three distinct groups, those brought about by pure gassing, the systemic effects of the vapour, and the effects on skin and mucous membranes. In the first instance death may occur from anoxaemia or toxic paralysis of the respiratory centre. Mild systemic effects include headache, giddiness, numbness, fits, diplopia, inability to accommodate, and vomiting; severe effects include signs similar to alcoholic intoxication with disorders of gait and co-ordination. Methyl bromide rapidly penetrates clothing, including leather and rubber, and has an irritant effect on skin and mucous membranes, producing burns characterized by enormous blisters containing sterile fluid. Depression and sleeplessness are frequent features of the convalescent period. Usually symptoms run parallel to the non-volatile bromine level in the blood which may be raised from a normal not exceeding 1·5 mg. per cent. to 10 mg. The bromine content in the cerebro-spinal fluid may also be raised from a normal of 1 mg./100 ml. to 15 or more mg./100 ml. Death, when it occurs, usually results from pulmonary oedema, and necropsy findings include acute purulent bronchitis, inflammation and oedema of the lungs, fatty degeneration of the ganglion cells of the brain and degenerative changes in the kidneys.

In 1940 Hunter, Bomford and Russell recorded four cases of poisoning by inhalation of methyl mercury iodide in a factory where fungicidal dusts were manufactured. The symptoms were entirely different from those of inorganic mercury poisoning, and resembled much more the nervous symptoms associated with methyl derivatives. They consisted of severe generalized ataxia, dysarthria, and gross constriction of the visual fields, while memory and intelligence were unaffected. Animal experiments showed that there was an intense and widespread degeneration of certain sensory paths of the nervous system, the peripheral nerves and posterior spinal roots being affected first, the posterior columns and the granular layer of the middle lobe of the cerebellum later.

Toxicity of Methyl Iodide

Methyl iodide is not extensively used and therefore few cases of poisoning have occurred. The only one reported in the literature is by Jaquet (1901). He recorded the case of a man, aged 30, who developed methyl iodide poisoning whilst employed in a dye-works. The symptoms were vertigo, diplopia, and ataxia, the patient swaying as if drunk. Chemical tests of the urine gave a strong reaction for iodine. When delirium and mania ensued treatment in a mental hospital was necessary. Mental dullness persisted and the man, unable to resume his work, was given the task of gate attendant, but up to the present time no case of methyl iodide poisoning has been recorded in Great Britain.

Methyl iodide is used as a half-way product in making certain other preparations. It is a colourless acrid liquid with a boiling point of 42·5° C. and melting point of −66·1° C.; it is 1·4 per cent. soluble in water and completely miscible with alcohol and ether. It is lachrimatory. At the factory where the present case occurred it was prepared by the interaction of dimethyl sulphate and sodium iodide in a still, the methyl iodide being collected over water in an open pan. The still was heated by means of a steam coil and the methyl iodide carried in an open vessel and poured into a carboy. It was an open method and undoubtedly exposed the workman to the fumes of methyl iodide, but at that time the risks were not appreciated and now the method is entirely enclosed.

Case Record

An account is given of the case of a worker on this process who developed the symptoms of methyl iodide poisoning.

A man aged 38 was employed in a chemical factory manufacturing methyl iodide. On December 12, 1940, he complained of giddiness, sleepiness and diarrhoea. There was some mental irritability accentuated by noise. He ceased work and was attended by a doctor, recovery occurring at the end of one week. The man remained well and resumed his work on March 10, 1941, but towards the evening of that day he developed vomiting and drowsiness. He saw the first-aid attendant who described him as looking pale and somewhat intoxicated.

The patient was admitted to hospital on March 11 when he was found to be drowsy and unable to walk. The speech was slurred and incoherent. There were nystagmoid movements of the eyeballs to the left and a slight degree of right internal strabismus. The fundi were clear. The muscles of the upper limbs twitched and the lower limbs were spastic. The abdominal reflexes were absent, but the deep reflexes and plantar responses were normal. The output of urine was reduced, only 1 pint being passed in 24 hours; it contained 9 mg. of combined iodine per 100 c.c., but no bromine or methyl alcohol. Lumbar puncture was performed on two occasions. A total of 20 c.c. of cerebrospinal fluid was withdrawn and found to be normal. There was no evidence of iodine, bromine or methyl alcohol on chemical examination. On March 13 vomiting, restlessness and incontinence occurred. From that time onwards the patient became comatose and the limbs flaccid, death taking place on March 18.

At the autopsy all the organs were seen to be congested, apart from the heart which was pale and flabby. There was broncho-pneumonia. Microscopic examination of the lungs showed areas of broncho-pneumonia and haemorrhages. The tissue around many small arteries was infiltrated with lymphocytes and plasma cells. Section of the brain showed congestion but nothing more. Chemical examination of the brain yielded a total of 6 mg. of combined iodine.

It will be seen that there is a striking resemblance between methyl iodide poisoning and poisoning with

methyl chloride and methyl bromide. This is only to be expected, but little confusion should arise in diagnosis since it is probable that it will be known to which of the three compounds the patient has been exposed. Further chemical examination of the urine, blood or cerebro-spinal fluid will reveal which of the halogens is present in excess.

Dimethyl sulphate from which methyl iodide is manufactured is a toxic substance but little difficulty is likely to arise here since the symptoms are entirely different. The fumes of this substance cause oedema of the mucous membranes which usually subsides in a period of 10 days; the symptoms include conjunctivitis, oedema of fauces and uvula, making swallowing difficult, and cough. There are no abnormal signs in the central nervous system and the mentality is unclouded.

Prevention

Everything depends on prevention. Legge (1934) emphasizes as one of the guiding principles of industrial medicine that all workmen must be informed of the dangers associated with the material used, and that they should not be left to discover their poisonous action by bitter experience. Due precautions have been taken with dimethyl sulphate, but methyl iodide has not always been regarded as dangerous, the product being ladled into containers by operatives with no gloves or masks. Workmen find it irksome to wear masks for long hours so that intervals of rest are essential. ' Festina lente ' must be the motto for employers who wish to have the maximum work performed without risking the lives of others.

Adequate ventilation is essential and in war-time black-out regulations may interfere with this. Irritant gases such as chlorine soon give warning of their presence, but when the poisonous substance is inodorous the first sign may be sudden loss of consciousness. Cages containing mice or birds placed at danger points should prove of assistance in the early detection of poison gas, but chemical tests are preferable and a ' Halide ' Detector Lamp can be employed to discover the presence of organic halogen compounds. The Department of Scientific and Industrial Research (1940) has issued a leaflet on the subject, but methyl iodide is not listed, and it is also to be noted that methyl bromide may give rise to acute symptoms in concentrations below that which can be detected by the lamp. Defects in the working of machinery may occur even in the apparently perfect factory. It is when these appear that the risk is greatest. Routine precautions are apt to be forgotten when the unexpected problem arises. More than once rescuers have themselves fallen victims by forgetting to remove the patient immediately symptoms have become manifest.

Treatment

Although lumbar puncture was performed for diagnostic purposes in the case recorded, it was hoped that it might prove to be a therapeutic measure combined with rectal injections of hypertonic magnesium sulphate. However, the cerebro-spinal fluid was not under increased pressure so that the treatment found little support from the theoretical point of view. Some halogen derivatives have caused neuritis. It was therefore decided to try vitamin B_1. Injections of massive doses were employed with no appreciable effect.

Summary

A case of methyl iodide poisoning is described. The signs and symptoms included nausea, vomiting, diarrhoea and oliguria, vertigo, slurred speech, visual disturbances, ataxia, tremor, mental irritability, drowsiness and coma.

Methyl iodide is prepared from dimethyl sulphate and the symptoms of exposure to the latter are recorded.

It is shown how closely methyl iodide intoxication resembles that of methyl bromide and methyl chloride, whilst confusion with acute alcoholism could occur in all three types of poisoning.

Some features of prevention and treatment are presented.

Acknowledgments

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THE SOLUBILITY OR DISTRIBUTION COEFFICIENT OF CARBON TETRACHLORIDE IN WATER, WHOLE BLOOD, AND PLASMA

BY

JOAN F. POWELL

From the Nuffield Department of Medicine, Radcliffe Infirmary, Oxford

In many clinical investigations, and particularly in those concerned with the toxicity of inspired industrial solvents, the solubility of the vapour in question in water, blood and plasma, is a factor of importance. To determine the solubility of vapours by experiments in vitro, it is necessary to devise an apparatus whereby a large and constantly renewed surface of liquid is brought into contact with a stream of the vapour-air mixture of given concentration. When the liquid is water this is easily achieved by passing a stream of small bubbles of the gas mixture through a column of liquid. In the case of blood and plasma this method is not practicable owing to the high viscosity of these fluids and their tendency to uncontrollable foaming. Several types of apparatus have been used in the past for overcoming these difficulties. To determine the solubility of chloroform, ethyl chloride and ethylene in blood, Nicloux (1929) used an apparatus devised by Scotti-Foglieni (1927) in which gas-air mixtures are passed over blood which is continuously agitated by vigorous stirring. To determine the solubility of carbon disulphide, McKee (1941) has used a system of two connected tonometers, one of which contains blood. The vapour-air mixture is passed through both tonometers for a few minutes, the system is then cut off from the gas supply, and rotated until equilibrium is reached.

To determine the solubility of carbon tetrachloride in blood and plasma, it was decided to construct an apparatus which maintained a constant moist gas-air flow through a rotating tonometer which contained blood or plasma. The apparatus (fig. 1) was designed by Dr. K. Mendelssohn, and a short description of it is given as an appendix.

Methods

The carbon tetrachloride-air mixtures were made up in gas cylinders under pressure. The concentration of carbon tetrachloride in the issuing gas varied between 500 and 5000 v.p.p.m., i.e. 6–32 mg. carbon tetrachloride per litre. In order that no condensation of carbon tetrachloride should take place, the partial pressure of vapour in the cylinders was kept below the saturation level. The maximum pressure allowable for the concentration required was calculated from

$$P(\text{max.}) = \frac{\text{Concentration of } CCl_4 \text{ in air saturated at } 20^\circ \text{ C.}}{\text{Concentration of } CCl_4 \text{ required in } CCl_4\text{-air mixture}} \text{ atmospheres}$$

Thus, in making up the 1000 and 500 v.p.p.m. cylinders, a pressure of 120 atmospheres is allowable, but at a concentration of 2000 v.p.p.m. this is reduced to 60 atmospheres, and at a concentration of 5000 v.p.p.m., to 24 atmospheres.

Analyses of the carbon tetrachloride-air mixture were made by passing a measured stream of gas into a known volume of toluene. The latter was contained in an absorption bottle with a sintered glass plate. An aliquot of the toluene solution was treated with pyridine and alkali (Habgood and Powell, 1944).

Water

Carbon tetrachloride-air mixture was bubbled through 40 to 60 ml. of distilled water contained in a gas absorption bottle with a sintered glass plate. Equilibrium was reached in ¾ hour with a gas flow of 50 ml. per minute. By connecting the gas supply to the outlet tube of the bottle, the liquid was transferred to a calibrated measuring cylinder. The latter had a ground glass neck, and contained a small measured volume of toluene, varying between 2 and 5 ml. The measuring cylinder was then stoppered and shaken at intervals for about 15 minutes until the toluene extraction was complete. The liquid was then allowed to settle, the toluene layer pipetted off and analysed for carbon tetrachloride by treatment with pyridine and alkali as before.

Blood and Plasma

Blood samples of 100–200 ml. were obtained from normal volunteers, oxalate or heparin being used as an anticoagulant. The haemoglobin content was measured by Haldane's carboxyhaemoglobin method. In a few cases the fat content was also determined by the method of Bloor (1928).

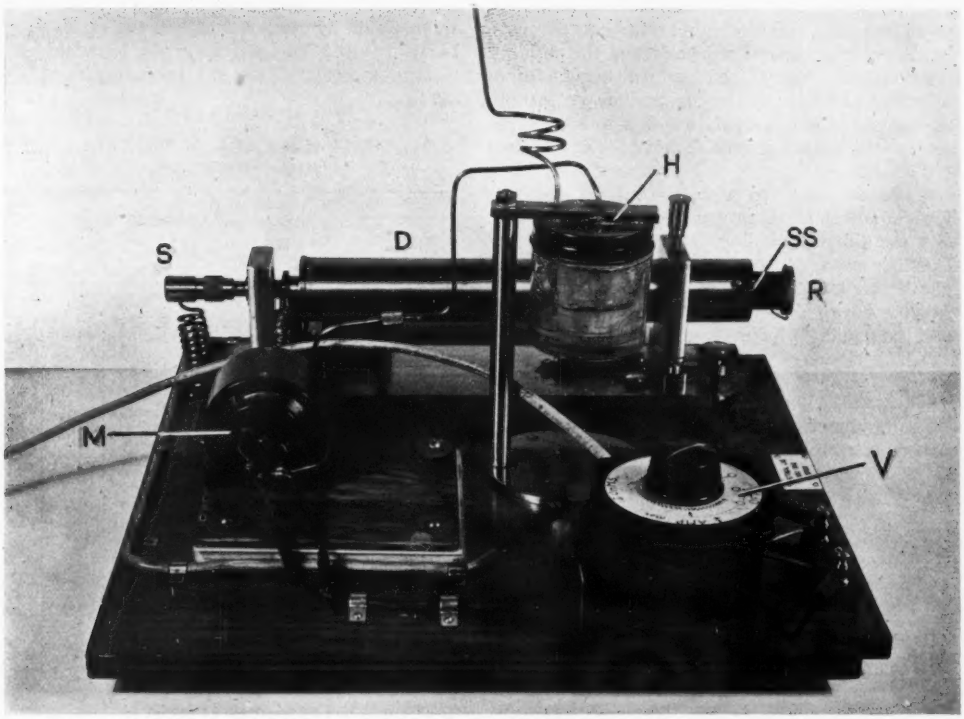

FIG. 1.—Equilibration apparatus.

FIG. 2.—Tonometer and sampling pipette.

To determine the amount of carbon tetrachloride absorbed from a given gas mixture, the water in the humidifier was first saturated with the gas mixture by passing through a slow stream, i.e. about 50 ml. per minute, for ½ hour. The tonometer was then loaded with 15–20 ml. of blood or plasma, which was generally enough for three analyses. The carbon tetrachloride-air mixture was then passed through the whole system at a rate of 250 ml. per minute, with the drum rotating at a speed of 100–120 revs. per minute. Blood samples were withdrawn after 1 hour, 1½ hours and 2 hours. For this purpose the pipette shown in fig. 2 was used—a modification of that described by McKee (1941). The pipette was first washed out with the gas mixture and then connected to the tonometer by means of the small ground glass joint. By tilting the tonometer slightly and applying a slight pressure from the gas cylinder to the flattened end, blood was forced to about midway up the bulb of the pipette. The taps were then turned off, and the tonometer disconnected. Pressure was then applied to the wide end of the pipette, and the blood forced into the bulb and up to the drawn out end. After adjusting the level, the blood was delivered into a stream distillation apparatus (Habgood and Powell, 1944) and its carbon tetrachloride content determined by extraction of the distillate with toluene and treatment of the toluene extract with pyridine and alkali.

It was found that, under the above conditions, 90–95 per cent. of the equilibrium concentration was achieved at the end of ½ hour, and 100 per cent. at the end of 1 hour. This performance could not be improved by increasing the gas flow to 500 ml. per minute. Decreasing the gas flow to 100 ml. per minute lengthened the time required for equilibrium to 2 hours.

It is seen that even with a relatively large gas flow, equilibrium takes considerably longer than with the simple bubbling arrangement which was used for water. A large proportion of the gas will take a straight course through the centre of the tonometer without coming into appreciable contact with the liquid.

Results

Water

The results obtained by equilibrating water with carbon tetrachloride-air mixtures at 20° C. and 37° C. are shown in fig. 3. The amount of carbon

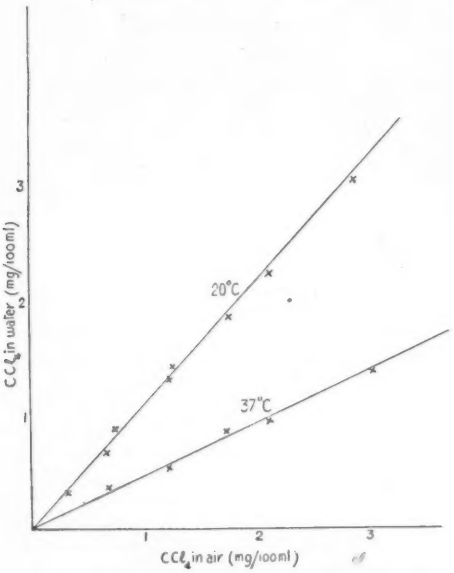

FIG. 3.—Solubility of carbon tetrachloride in water at 20° C. and 37° C.

tetrachloride absorbed is directly proportional to its concentration in the gas, the Ostwald solubility, or distribution coefficient, i.e. concentration in the liquid/concentration in the gas, being 1·07 at 20° C. and 0·46 at 37° C.

It is interesting to note that a saturated aqueous solution of carbon tetrachloride at 20° C. contains 80 mg. per 100 ml. Here the concentration of carbon tetrachloride in the gas phase is 76 mg. per 100 ml., so that the distribution coefficient is 1·05. Similarly at 37° C. we have found the solubility of carbon tetrachloride in water to be 68 mg. per 100 ml., with a concentration in the gas phase of 152 mg. per 100 ml., so that the distribution coefficient is 0·45. These values practically coincide with those found at the lower end of the scale, so it is likely that the solution of carbon tetrachloride in water obeys Henry's law at all concentrations.

TABLE 1A

SOLUBILITY OF CARBON TETRACHLORIDE IN BLOOD AT 20° C. AND 37° C.

Donor	CCl_4 in air mg./100 ml. (0·64 mg./100 ml. = 1,000 v.p.p.m.)	CCl_4 in blood		C blood/C air	
		20° C.	37° C.	20° C.	37° C.
1. Haemoglobin, 112%	0·33	1·72	—	5·2	—
	0·65	3·10	1·59	4·8	2·4
	1·32	6·67	3·07	5·1	2·3
	2·01	10·2	4·90	5·1	2·5
	2·86	14·3	7·06	5·0	2·5
2. Haemoglobin, 90%	0·34	1·38	—	4·0	—
	0·69	2·58	1·37	3·8	2·0
	1·20	4·54	2·42	3·7	1·9
	3·40	12·8	6·20	3·8	1·8
3. Haemoglobin, 95%	0·64	2·87	—	4·6	—
	1·10	5·25	2·35	4·8	2·1
	2·36	10·5	—	4·4	—
	2·77	13·4	5·9	4·8	2·1
4. Haemoglobin, 90%	0·33	1·76	—	5·3	—
	0·65	3·27	1·58	5·0	2·4
	1·32	6·67	3·07	5·1	2·3
	2·01	10·2	4·90	5·1	2·5
	2·86	14·3	7·06	5·0	2·5

TABLE 1B

SOLUBILITY OF CARBON TETRACHLORIDE IN BLOOD AT 20° C.

Donor	CCl_4 in air, mg./100 ml. (0·64 mg./100 ml. = 1,000 v.p.p.m.)	CCl_4 in blood, 20° C.	C blood/C air, 20° C.
5. Haeomglobin, 108%	0·34	1·40	4·1
	0·63	2·32	3·7
	1·20	4·80	4·0
6. Haemoglobin, 90%	0·34	1·28	3·8
	0·61	2·32	3·8
	1·27	5·60	4·4
	3·21	13·0	4·1
7. Haemoglobin, 96%	0·75	2·70	3·6
	1·22	4·40	3·7

As is seen from Tables 1A and 1B, the distribution coefficient of carbon tetrachloride between blood and air varied between 3·6 and 5·2 at 20° C. and between 1·8 and 2·5 at 37° C. Fairly constant values, within the limits of experimental error, were obtained from each blood sample. From the small amount of data obtained (Table 2), it seems likely that a correlation exists between the amount of carbon tetrachloride absorbed and the blood fat, although more experimental data is needed to establish this point.

There does not, however, appear to be any direct correlation between the red cell content and the amount of carbon tetrachloride absorbed, cf. cases

TABLE 2

SOLUBILITY OF CARBON TETRACHLORIDE IN BLOOD SPECIMENS OF DIFFERENT FAT AND HAEMOGLOBIN CONTENTS

CCl_4 in air (mg./ 100 ml.)	CCl_4 in blood(mg./ 100 ml.)	C blood/ C air	Haemo-globin (% Hal-dane)	Blood fat (mg./ 100 ml.)
0·60	1·95	3·3	90	384
	2·53	4·2	98	550
	2·15	3·6	108	540
3·44	12·5	3·6	92	394
	12·0	3·5	90	384
	15·5	4·5	94	500
1·32	4·40	3·3	90	384
	5·10	4·0	92	500

1, 4 and 7 in Tables 1A and 1B. Furthermore, blood obtained from a patient with hypochromic anaemia and a haemoglobin of 60 per cent. absorbed 7·03 mg. carbon tetrachloride per 100 ml. in contact with a gas mixture containing 1·32 mg. per 100 ml., and 3·40 mg. per 100 ml. in contact with a mixture containing 0·70 mg. per 100 ml. This gave high values for the distribution coefficient of 5·2 and 4·9 respectively.

The distribution coefficient between plasma and air varied between 6·2 and 10·7 at 20° C.

A comparison of the distribution coefficient of whole blood with plasma obtained from the same specimen is given in Table 3 and in fig. 4. These results indicate that the amount of carbon tetrachloride absorbed by the red cell is almost negligible. In two cases, small negative values were calculated from the experimental data—this was, of course, due to a too high value for the plasma, associated with a too low value for the corresponding whole blood. In the last colume of Table 3, theoretical values have been calculated for the carbon tetrachloride content of the red cells, assuming that the amount absorbed depends only on their water content.

TABLE 3

SOLUBILITY OF CARBON TETRACHLORIDE IN WHOLE BLOOD AND PLASMA AT 20° C.

Concentration of CCl_4 (mg./100 ml.)			Haema-tocrit	CCl_4 in red cells (mg./100 ml.)	
Air	Blood	Plasma		Calculated from ex-perimental results	Calculated from water content
1·32	5·10	8·20	41	+0·3	0·8
0·69	2·58	4·88	43	−0·5	0·4
1·20	4·54	7·70		+0·4	0·7
0·33	1·76	2·94		+0·1	0·2
0·65	3·27	6·00	42	−0·3	0·4
1·32	6·15	10·0		+0·6	0·8
2·36	15·0	25·0		+1·2	1·4
0·33	1·72	2·72		+0·7	0·2
0·65	3·10	5·66	50	+0·6	0·4
1·32	6·67	12·2		+1·1	0·8

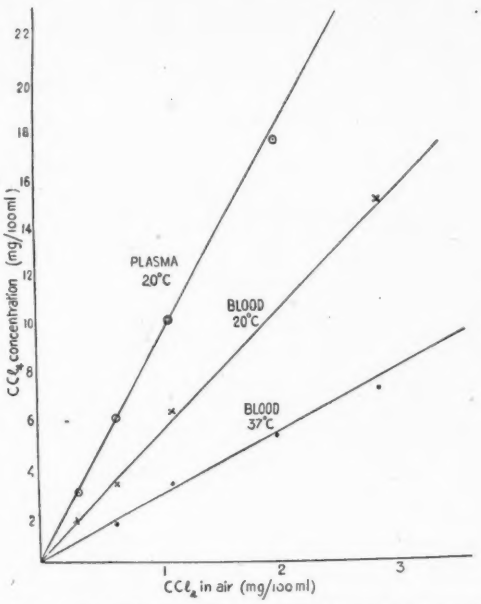

FIG. 4.—Solubility of carbon tetrachloride in whole blood at 20° C. and 37° C., and in plasma at 20° C.

It was found that prolonged contact, i.e. up to 4½ hours, with the gas mixture did not increase the amount of carbon tetrachloride absorbed by a given blood specimen, which practically removed the possibility that passage of carbon tetrachloride into the red cell is merely a slow process. Further, it was found that totally haemolysed blood, i.e. blood which had been kept at 0° C. for 2–3 weeks and shaken vigorously at intervals, absorbed the same amount of carbon tetrachloride from a given air mixture as it did before this treatment—which establishes the non-affinity of the red cell constituents for carbon tetrachloride.

An interesting result was obtained when the relative proportion of plasma to red cells was increased by diluting 1 volume of blood with 1 volume of plasma from the same specimen. When the whole blood, 'diluted' blood and plasma were equilibrated with an air mixture containing 3 mg. carbon tetrachloride per 100 ml., concentrations of 11·7, 15·0 and 20·0 mg. carbon tetrachloride respectively were found. The concentration in the 'diluted' blood was almost exactly midway between blood and plasma.

Discussion

The results obtained for water do not require further comment. In the case of blood and plasma, it is rather surprising to note the very small affinity of the red cell for carbon tetrachloride—in fact, the experimental data indicate that the small amount of carbon tetrachloride actually absorbed by red cells can be accounted for by their water content. It is

interesting to compare these results with those obtained by Nicloux (1929) for chloroform. This author finds that the distribution coefficient of chloroform between water and air varies between 15 and 4 in the temperature range 13° C. to 40° C., and the air-concentration range 5–12 mg. per 100 ml. The distribution coefficient between human blood and air was found to be 20 at 13° C., 16 at 30° C., and 10 at 37° C., and ox blood gave similar results in the same concentration range. Unfortunately no results are available for a lower range of concentrations comparable with those used in the present study for carbon tetrachloride. Since, however, from Nicloux's results, the distribution coefficient at a given temperature is practically constant throughout the range investigated, it is likely that the same relationships hold at lower concentrations.

In the case of serum obtained from ox blood, Nicloux has found values for the distribution coefficient to vary between 20 and 8 in the temperature range 13° C. to 27° C. It therefore appears that, in marked contrast to their behaviour with carbon tetrachloride, red cells absorb chloroform to approximately the same extent as does serum. This is supported by the experiments of van Dessel (1923), who added small amounts of chloroform to oxalated human blood, and recovered 52 per cent. of the amount added in the plasma, and 48 per cent. in the red cells. In blood taken from patients under chloroform anaesthesia, however, it should be noted that the same investigator found approximately 70 per cent. of the chloroform in the plasma. Scotti Foglieni (1930) found that the haemoglobin content of blood is of primary importance in determining the amount of chloroform and of ethyl chloride which it will absorb. This is supported by the evidence that suspensions of pure haemoglobin in water absorb quantities of chloroform and ethyl chloride proportional to the amount of haemoglobin present. The presence of red cell envelopes and stroma does not influence these results. The same author also states that serum free from traces of haemoglobin gives the same solubility coefficient as pure water—a very surprising observation in view of the very great solubility of chloroform in fatty media.

The results obtained in the present study indicate that the plasma fat is the determining factor in the absorption of carbon tetrachloride, and that the haemoglobin content reduces the absorption in a purely mechanical way, i.e. by decreasing the relative amount of plasma. Carbon tetrachloride is chemically a relatively inactive compound compared with chloroform, and its non-affinity for haemoglobin may be thus accounted for. It is true that red cells contain quite large amounts of 'lipoid' substance, which might be expected to dissolve chlorhydrocarbons readily. This 'lipoid' material is not, however, in the form of simple fats, but consists mainly of phospholipoids (Macey, 1938), in which carbon tetrachloride may be relatively insoluble. The observation of Scotti Foglieni that the red cell constituents other than haemoglobin do not influence the absorption of chloroform, is of relevance in this connection.

Summary

The distribution coefficient of carbon tetrachloride between water and air is 1·04 at 20° C. and 0·46 at 37° C. The air concentrations used varied between 0·33 and 3·44 mg. per 100 ml. (500–5,000 v.p.p.m.). The 'distribution coefficient' is defined as concentration in the liquid/concentration in the gas.

The distribution coefficient between blood and air is constant in a given specimen throughout the above concentration range. The coefficient in a series of specimens varied between 3·6 and 5·2 at 20° C. and between 1·8 and 2·5 at 37° C.

The amount of carbon tetrachloride absorbed by the red cell appears to be negligible.

The small quantity of data obtained suggests that the amount of carbon tetrachloride absorbed from a given air mixture is proportional to the plasma fat concentration.

Acknowledgments

I wish to thank Dr. K. Mendelssohn for his help in designing the equilibration apparatus, and Miss Sheila Habgood for assisting with the blood fat estimations.

APPENDIX

The Equilibration Apparatus (see Fig. 1)

K. MENDELSSOHN

The gas mixture is first passed through a flowmeter and then enters the humidifier (H). This consists of two concentric compartments separated by a cylinder of wire netting covered with a few layers of gauze bandage. The latter is kept moist by capillary attraction. The gas mixture entering the inner compartment has to pass the moist screen before it can reach the outlet tube which opens into the outer compartment. From the humidifier, the gas passes into the rotation drum (D), which houses the tonometer (see fig. 2). Junction between the stationary inlet tube and the rotating drum is effected by a close but not tight fitting metal sleeve (S), which is kept in position by the coiled inlet tube. Since the flow resistance of the whole arrangement is kept very low, the leak through this junction is negligible. The tonometer is kept in position, its flattened end pressed into the drum by the ring (R) and the springs (SS). From the tonometer, the gas mixture escapes into the atmosphere. The small motor (M) rotates the drum through a worm-drive, and its speed is controlled by the variable resistance (V). The component parts are mounted on a plywood board, which is fitted into an ordinary incubator, the leads from the accumulator and the gas supply being passed through a hole in the top of the incubator. A detailed description of the instrument and its performance will be given later.

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THE INCIDENCE OF SEPSIS IN INDUSTRIAL WOUNDS

BY

R. E. O. WILLIAMS AND E. H. CAPEL

From the Medical Research Council Wound Infection Unit, Birmingham Accident Hospital, and the Medical Department, Joseph Lucas Limited, Birmingham

In this paper we report an investigation of the prevalence of sepsis among the workers in an engineering factory, and attempt to estimate the risk of sepsis in the wounds inflicted there.

Official figures for sepsis in a selected group, namely reportable accidents (i.e. those resulting in more than three days' absence from work), are collected by H.M. Inspector of Factories and they have been discussed by Henry (1943). These figures do not, however, directly measure the incidence of sepsis in wounds, since the number of cases reported as septic is set against the total number of reportable accidents, which includes not only wounds but also eye injuries and closed injuries (hernia, closed fractures, muscle strains, etc.) which are not at risk for the development of sepsis. Furthermore, the figures do not include wounds, serious enough to lead to absence from work from the day of injury, that become septic after notification. The official sepsis rate, therefore, gives the number of workers who are put off work on account of sepsis that is already present at the time of notification, and not the total number whose wounds become septic, nor the number whose return to work is delayed by sepsis. Septic wounds that do not lead to four days' absence from work are not included at all.

To demonstrate the implications of the published figures we have analysed the records for 1944 from one factory engaged in light engineering and employing an average of 7430 workers. The inquiry cannot readily be extended to other factories, because it is made possible, in this case, by a relatively rare combination of circumstances: the medical department keeps record cards for each individual attending the surgery, so that the history of each wound is traceable; almost all the wounds that require hospital treatment are sent to one hospital (the Birmingham Accident Hospital) where their records are again available; and we have the data gathered in a close clinical and bacteriological study of some 280 of the wounds treated in the surgery of the same factory in autumn of 1943 (the factory A of Clayton-Cooper and Williams, 1945).

Reportable Accidents

During 1944, 266 accidents led to more than three days' lost time (Table 1). The 'sepsis rate,' as calculated by the Factory Inspectorate, was 27·4 per cent., i.e. 73 of the 266 workers were put off work on account of sepsis. However, 123 of the 266 workers had no open wound. Of the remaining 143, 73 (51 per cent.) had septic lesions, in most cases probably arising from trivial wounds (see below) which would not have led to loss of time unless the sepsis had developed; and 63 had wounds which were not septic but were severe enough to lead to absence from work from the day of injury. The notes of the remaining seven patients could not be traced.

TABLE 1

ANALYSIS OF ACCIDENTS REPORTED IN 1944

(*a*) Total number of accidents reported to Factory Inspectorate	266
(*b*) Number of accidents reported as 'septic'	73
(*c*) Number reported as 'clean wounds' ..	63
(*d*) Number of accidents not resulting in wounds (eye injuries, closed fractures, sprains, hernia, etc.)	123
(*e*) Wounds, notes not traceable	7
(*f*) *Percentage of all accidents* (a) *reported as 'septic'*	*27·4*
(*g*) *Percentage of accidents other than those in group* (d) *reported as septic*	*51·0*
(*h*) Number of wounds in group (*c*) that developed sepsis after reporting ..	8
(*i*) I.e. total number of wounds septic at some time	81
(*j*) *Total percentage of all reported accidents* (a) *that were septic at some time* ..	30·4
(*k*) *Total percentage of accidents other than those in group* (d) *that were septic* ..	*56·6*

Sepsis may develop in a wound while it is under treatment at the surgery, or it may develop in a hitherto untreated wound and induce the worker to attend the surgery. Further analysis of the factory records shows that, of the 73 wounds reported as septic, 66 (90 per cent.) were already septic when they were first seen at the surgery, and only seven had become septic, and therefore reportable, while under treatment. Eight of the 63 initially clean serious wounds later developed sepsis.

Non-reportable Accidents

If, instead of studying the reportable accidents, we turn to the data for all the wounds presented to the factory surgery, we obtain very similar proportions (Table 2). In 1944, there were 8101 first attendances at the surgery by workers with cuts, burns, abrasions, etc.; of these 1050 (13 per cent.) were already septic at this first attendance. Inquiry of 230 randomly selected patients attending the surgery with such septic lesions revealed that in 76·5 per cent. the lesion had arisen from a trivial

wound which the patient had noticed at the time of injury, but for which he did not then have treatment; some sign of an antecedent injury was visible in a further 13·5 per cent.

TABLE 2

ANALYSIS OF SURGERY RECORDS FOR 1944

Total attendances at surgery with cuts, burns, abrasions, and septic lesions	8101
Attendances with non-septic wounds	7051
Attendances with lesions septic at first attendance	1050
Estimated number of initially clean wounds that became septic before healing (*5 per cent. of total*)	*353*

Clayton-Cooper and Williams (1945) found that the incidence of sepsis developing in initially clean wounds attending the surgery (in the autumn of 1943) was in the region of 5 per cent. The incidence is unlikely to have risen since, and may well have fallen, because in the interval a number of improvements were made in the surgery routine. If, however, we take the figure of 5 per cent. to be applicable to the 7051 fresh wounds seen at the surgery in 1944, we would expect 353 to have become septic under treatment; in fact we note that approximately three times as many wounds as this (1050) were observed to come to the surgery already septic.

Studying the proportions of septic and clean wounds that would be recorded by the Factory Inspectorate because they led to loss of time, there is the same excess in the percentage of wounds septic when first seen over the percentage becoming septic while under treatment. Of 7051 workers with wounds seen at the surgery soon after the injury 63 (0·9 per cent.) had to be put off work immediately, and a further seven (0·1 per cent.) were put off later because severe sepsis developed in the wound while it was under treatment. Thus 1 per cent. of the workers with wounds presented to the surgery at the time of injury were put off work at some time. On the other hand, of the 1050 workers with wounds that were septic when first seen, 6·3 per cent. (66) were off work more than three days; this proportion is more than six times that for the clean wounds.

Untreated Wounds

The facts presented suggest that the workers were, in many instances, failing to attend the surgery with small cuts unless the development of sepsis in the wound made attendance necessary, and it seemed desirable to attempt to estimate the risk of sepsis in the trivial untreated wounds. At Professor Bradford Hill's suggestion we accordingly tried to make a count of all the wounds inflicted in the factory on one day.

A questionary form (fig. 1) was given to each charge-hand (responsible for 10–30 workers) and he was told of the aim of the investigation and the method of completing the form; we defined an injury as any lesion that drew blood from the skin. Thinking that some workers might fail to report injuries if they thought that disciplinary action would be taken against those who had not obeyed the standing rule to present all injuries for treatment, we did not ask for names or check numbers. The questionary was completed in the last half-hour of the day's work, each worker reporting the number of injuries sustained in the day. Owing to the administrative structure of the factory, it was possible to survey only 'production workers,' i.e. those directly concerned with production and not the maintenance and office staff, whose injuries contributed to the totals given in Table 2. From

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IMPORTANT

In connection with the investigations being carried out by the Medical Research Council on the prevention and treatment of smaller injuries occurring in factories, it is desired to make a count of all injuries, serious or trivial, occurring on one particular day.

This form is being issued to all charge-hands, who are asked to carry out the following instructions accurately.

Instructions.

1. Using a separate square for each worker fill in the number of injuries, serious or trivial, sustained by him or her during the single day.
2. If a worker sustains no injuries put 0. This is most important.
3. Take the count during the last half hour of the day's work.
4. Return the form immediately to your superintendent.

Department .....

Read the following notes:-

Count all injuries, however trivial, sustained at work during the single day only.

It does not matter whether the injury was treated or not, count it.

Don't forget to count yourself.

This investigation is concerned with research into means of prevention and treatment only. No names or check numbers are to be given.

FIG. 1.—Questionary form used for census of injuries.

fear of causing too great a loss of working time, we have only taken two counts at the factory from which we gathered our main data, but comparable investigations have been carried out in two similar factories. We have estimated the risk of sepsis in the wounds by relating the daily number of untreated wounds, derived from the census, to the average daily number of wounds presented to the surgery already septic, on the assumption that the great majority of the latter arise from trivial wounds not treated on the day of injury.

With the many limitations, and with the additional difficulties that many of the smallest wounds may not be noticed by the worker, and that some of the septic lesions certainly do not arise from wounds such as are counted in the census, it is evident that our results can give only the very broadest indication of the rate of wounding and the risk of sepsis. Nevertheless, we feel that they do enable us to get a clearer idea than was previously possible of the risk of sepsis in small untreated wounds.

The results of the census at three different factories are summarized in Table 3. The striking feature is the comparatively large proportion of the workers who admit to some sort of injury on the day of the census—6–16 per cent. of the number questioned—and the relatively small proportion of the injured workers who attend the surgery. Many of the injuries are doubtless extremely small, e.g. puncture wounds made by swarf, and might therefore be considered unimportant by the worker. To assess the sepsis rate in the wounds, we have calculated the average number of wounds that were presented to the surgery already septic in the 3-month period around each census day, and the last row in Table 3 shows the estimated daily number of such wounds per 100 wounds untreated at the time of injury, derived from the census. If our premises are correct, it appears that the incidence of sepsis in the untreated wounds is unlikely to have been more than 1–2 per cent.

The agreement of the figures from the three factories shown in Table 3 is noteworthy and lends some weight to the results. It must be added, however, that an attempt to repeat the census in a machine shop of a different firm was not so satisfactory; the figures derived from one week's questioning differed widely from those obtained in a subsequent week, though both agreed in revealing a very large number of untreated injuries. It is probable that the discrepancy was due in part to a different interpretation of the term ' cut ' in the two weeks, and in part to the fact that the first week, during which the lower incidence was recorded, fell soon after the Christmas holiday, when the shop was not working to capacity.

Discussion

The implications of the ' sepsis rate ' given by the Factory Department must be made clear. The statement that the rate was 27 per cent. for this factory does not mean that sepsis supervened in 27 per cent. of the wounds inflicted in the factory, or even in 27 per cent. of those leading to loss of more than three working days. It means simply that, of the 266 workers put off work more than three days on account of accidents, 73 were put off because of sepsis. In fact, 143 workers had accidents that were not of the sort that could show sepsis; another 63 had serious wounds at risk for sepsis, but they were notified before the sepsis could have developed, and sepsis did develop later in the wounds of eight of them.

A sepsis rate of 27 per cent. is higher than the average for the country, but this does not necessarily mean that an undue proportion of the wounds in this factory developed sepsis. This is one possible explanation, but another might be that the work done was of the sort that is likely to lead to a large number of trivial injuries but to few serious accidents leading to absence from work from the day of injury. To use the figures for the incidence of sepsis to assess the average risk of sepsis to wounds inflicted in the factory demands a knowledge of the total number of wounds that were inflicted, and the proportion of them that were serious. We estimate that about 10 per cent. of the workers injure themselves each day, although, during the whole of 1944, only 63 of the wounds were severe enough to lead to absence for three days from the day of injury.

We would not lay great stress on the numerical

TABLE 3

RESULTS OF CENSUS OF INJURIES

	Factory 1*		Factory 2		Factory 3		
Date of census	Sep. 27, 1944	Jan. 1, 1945	Oct. 25, 1944	Nov. 24, 1944	Dec. 12, 1944	Dec. 13, 1944	Dec. 14, 1944
(*a*) Number of workers questioned ..	2419	2878	2424	2365	158	161	160
(*b*) Number recording an injury † ..	200	173	171	143	22	26	26
(*c*) I.e. *number of injured per* 100 *workers* (100*b*/*a*)	*8·3*	*6·0*	*7·1*	*6·1*	*13·9*	*16·2*	*16·3*
(*d*) Number of attendances at surgery with a fresh wound	19	28	43	34	4	10	12
(*e*) I.e. *number attending per* 100 *injured* (100*d*/*b*)	*9·5*	*16·2*	*25·2*	*23·8*	*18·2*	*38·5*	*46·0*
(*f*) Number of injured workers not attending surgery	181	145	128	109	18	16	14
(*g*) Number of septic wounds presented per day, average of 3-month period	3·4	3·5	1·4	1·4	0·3	0·3	0·3
(*h*) I.e. *number of septic wounds presented per* 100 *injured workers not attending on day of injury* (100*g*/*f*)	*1·9*	*2·4*	*1·1*	*1·3*	*1·7*	*1·9*	*2·1*

* This is the factory upon which the main report is based, and all the figures from it in this table refer to 'production workers' only. In the other factories, the census included all the workers.

† The figures refer to the number of workers reporting an injury; the average number of injuries per injured worker was 1·2.

results reported; they are derived from a study of one factory only and, though great care was taken, the methods of obtaining them are certainly open to some criticisms. There can be no doubt, however, on two points: (*a*) trivial wounds are extremely common in the factory, and (*b*) the great majority of the septic lesions that are seen in the surgery are septic when first seen, and probably arise from trivial injuries untreated at the time of the accident. Owing to the great number of such injuries, the number of septic wounds, though apparently large, represents but a small proportion of the untreated wounds. Our data suggest that about 5 per cent. of all the injuries treated in the factory surgery on the day of injury develop sepsis, and that the corresponding proportion for the wounds that are not so treated is about 1–2 per cent. In view of the methods by which the figures were obtained, we do not feel justified in drawing any conclusions as to the value of surgery treatment from this small difference. It certainly cannot be used as evidence that the surgery treatment is harmful, because the wounds that are presented to the surgery are on the average likely to be more serious than those that are left untreated, and it is probable that the serious wounds are more liable to become septic than the trivial. In the series observed by Clayton-Cooper and Williams it was, on the whole, the more serious wounds that became septic; and the higher sepsis rate among the large wounds sent to hospital, compared with those treated in the factory surgery, probably reflects the same difference.

That sepsis is not more common among the untreated wound does, however, raise a question as to the importance of the customary exhortation towards early surgery treatment for all wounds. It is evident from our census figures, and from the experience of anyone who has examined the hands of metal workers, that the great majority of small cuts do not become septic, even when untreated. Furthermore, the surgery treatment of *all* trivial cuts—even if it were possible to induce the workers to attend—would demand a five- to ten-fold increase in surgery staff and accommodation, and would lead to an intolerable loss of working time. Whether it would materially reduce the incidence of sepsis is doubtful. It seems clear that, to some extent, the individual worker must decide whether a small cut requires surgery treatment. We feel that much of the effort spent in the past in exhorting the workers towards early and regular attendance at the surgery with every cut, might be more profitably employed in devising methods of preventing the injuries that allow the entry of bacteria into the tissues, as for instance by designing machines that do not require the worker to handle sharp swarf. It is not our intention to suggest that the surgery services are redundant; rather would we emphasize that the provision and efficient running of a surgery is not, by itself, enough to solve the problem of industrial sepsis, and that the medical services cannot evade responsibility for the still high incidence of sepsis by the plea that the workers do not attend for treatment sufficiently soon. Sepsis should be regarded as an occupational hazard of the same sort as, for instance, lead poisoning, and its prevention lies not only in the efficient treatment of the workers at greatest risk—those with small wounds—but also in the prevention of those wounds.

Summary and Conclusions

Analysis of the records for 1944 from one engineering factory employing 7430 workers reveals that there were in all 266 accidents leading to more than three days' absence from work. In 73 cases, the worker was put off on account of sepsis, i.e. the 'sepsis rate' as calculated by the Factory Inspectorate was 27·4 per cent. However, only 143 of the accidents led to wounds at risk for sepsis, and of these 51 per cent. were septic when reported. Ninety per cent. of these septic wounds were septic when first seen at the surgery.

In the same year there were 8101 first attendances at the factory surgery: 7051 with fresh wounds, etc., and 1050 with septic lesions. We can estimate that about 350 of the 7051 fresh wounds probably developed sepsis while under treatment.

Using the data from a census taken to determine the total number of wounds inflicted in the factory, we can estimate that between 6 and 16 per cent. of the workers in the factory suffered an injury on any one day, but that only 9–25 per cent. of the injured workers attended the surgery. We estimate that the incidence of sepsis among the untreated wounds was of the order of 1–2 per cent.

Though the great majority of the cases of sepsis that lead to loss of time occur as a result of the trivial injuries not treated at the time of the accident, the incidence of sepsis in such wounds, in proportion to the very great number that occur, is low, and it does not seem that the elimination of industrial sepsis is to be achieved solely by increasing the aseptic and antiseptic efficiency of the factory surgery. Increasing the proportion of injured workers who attend the surgery might have some effect, but it would be difficult to achieve, and if achieved, it would be uneconomic. Serious attempts must also be made to prevent the occurrence of the trivial injuries that are largely responsible for the sepsis.

Acknowledgments

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The census could not have been carried out without the help from many members of the staff and from the charge-hands in the factories surveyed, and to all of them our thanks are due.

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RESETTLEMENT AND INDUSTRIAL MEDICINE

A major problem facing the country and industry to-day is the resettlement of men and women discharged from the Forces. This has already had repercussions on health, frequently of a psychological character, which need careful thought and study. The industrial medical officer has therefore additional responsibility to help place these men and women in work which must be not only within their physical capacity but of such a type that it produces a feeling of settlement and contentment. Recent emphasis on rehabilitation, and its liberal definition to include all types of disablement, should have done much to prepare him for this responsibility; but certain additional factors need emphasis and in this connexion a recent publication by PEP [1] is of much importance. 'With war at an end,' says PEP, 'Government schemes for resettling some four million ex-servicemen and women in civilian life within a matter of months will be put to the test. The sheer magnitude of this operation, coupled with inter-industrial transfers, and touching as it does almost every family in the country, scarcely calls for emphasis; although it is doubtful if the Forces themselves or the general public fully appreciate the many difficulties, particularly those of a psychological character, which are likely to arise.'

One aspect of resettlement concerns the disabled ex-serviceman and plans are prepared, through the Disabled Persons (Employment) Act of 1944, for the medical profession to take an active part in this. The responsibility of the industrial medical officer within this narrower sphere is gradually becoming more obvious, both in his own organization and, it may be, as adviser to local committees. But as PEP points out the distinction between the fit and the disabled is in many respects an artificial one; fit and disabled alike need not only the service of efficient resettlement machinery, but also sympathetic help and advice from officials who like and understand their fellows. 'Fundamentally, resettlement is an exercise in the art of treating people as free human beings, not arbitrarily classified units.'

The psychological background to the whole problem needs elaboration and this aspect is of fundamental concern to the medical profession both inside and outside industry.

Thousands of men and women at the outset of their war service were ordinary civilians, indistinguishable in their habits and outlook from those left behind to work in factories, shops or Ministries. But from the day they joined their units they entered into a process of change. The needs of the time demanded that they should be bodily fit and able to endure physical and nervous strain; that they should become a disciplined force carrying out orders on the word of command; that their minds should turn from the intimate little problems of peace to the wider issues of the survival of freedom and the defeat of the enemy. Training and good food improved their physique so that lanky adolescence and the seediness of a sedentary life gave place to an appearance of ruddy toughness. Under the direction of officers and N.C.O.s, they learned to work as a team and to do without question what they were ordered. They developed a sense of corporate responsibility towards their comrades, and, as this new relationship strengthened, older ties weakened, and they began to lose touch with civilian life as they had known it.

With the end of the war came an immediate and instinctive desire for release, but unwinding the machine has been in many ways a more delicate and difficult operation than the call-up. The returning serviceman has to accustom himself not only to changes in his home and in the community generally, but also at his job.

If he goes back to his old job he will find that he has missed years of industrial development. His fellow-workers will be familiar with the new techniques, but he may have a long way to catch up. The ease with which he does so will depend on the extent to which the sympathetic consideration of his employers has been applied to meeting the needs of resettlement. If, at the outset, he is made to feel awkward, his whole future career may be threatened. He will become resentful, and the rift between him and his mates will be deepened. He will feel that his war experience belittles the smaller day-to-day problems of industry. He will know in his heart that he has been caught up in fundamental issues; and he will be right.

Dissatisfaction may also arise in the case of those men of good education who had, or would have, entered industry in peace time to learn a practical profession for which a university qualification was not necessary. They will now have little background knowledge and they may have to start completely afresh, learning the elements of their particular industry.

Then there will be a change from an active outdoor life to indoor routine with hours which must be kept. This may be found irksome at first, but in many cases the diversions and amenities of town life will influence the returning soldier and this unrest should be short-lived. Undoubtedly there are men who feel restless, and some single men at any rate might be happier if they were regarded as mobile workers; industry now has a place for them which was not adequately recognized before. There will also be men who feel that they could never spend their lives working indoors, and they could be encouraged to enter the building industry or agriculture. It is of the utmost importance that these men re-entering industry should, wherever possible, be able to take up the employment they prefer in the light of the new experience and skills they have gained during their years of war service; for a man is most efficient when he is doing a job he likes.

Schemes to help solve some of these underlying difficulties have been evolved by the Government. Pre-release schemes have been instituted by the Ministry of Labour collaborating with the educational branches of the various services; there are 360 Resettlement Advice Offices throughout the country 'with cream walls, gay paintwork, comfortable chairs, and a reception counter of pleasing design'; and Appointments Offices and the Employ-

[1] *Resettlement.* Planning Broadsheet No. 239, Sept. 1945. PEP (Political and Economic Planning), 16 Queen Anne's Gate, London, S.W.1.

ment Exchange Service put those seeking employment into touch with would-be employers. But most of the success of official planning will depend not so much on office background but on the character and personality of the individuals who operate the scheme; they should themselves be specially selected, and should have understanding of human beings, aptitude for interviewing, and some knowledge of industry. The Civil Resettlement Units of the Army, set up in May of this year, help repatriated prisoners of war discharged or released from the service. They have already done much to prevent psychiatric casualties in the change-over period. The scheme is voluntary, but has been well supported; particular attention is paid by experienced psychiatrists to the needs, and fears, of the individual; simple schemes of vocational guidance of different types have been instituted; the officers who staff these Units are themselves well-informed as to the needs of local factories; and they are accustomed to man management. It is good to know that the medical profession has played a leading part in this venture.

Only a small proportion of ex-servicemen present problems in mental health during the resettlement phase. The majority appear to be returning to peace-time work with little trouble. It would be a mistake to assume, for example, that every ex-prisoner of war is a potential patient for the psychiatrist, or that the majority of returning servicemen are unhappy or depressed by civil life. But difficult cases are occurring regularly, and in organizations where industrial medical officers are established there is much scope for a new type of preventive medicine. Pre-placement medical examination is an essential part of industrial medical practice and should include all those returning from service with the Forces, no matter what their job is to be. There is already evidence that these examinations reduce labour wastage and increase industrial efficiency and at the same time decrease absence due to sickness. To help him the doctor in the factory should be provided by the Ministry of Labour with the relevant health history and, in the case of disabled workers, with the report and recommendations of the Disablement Rehabilitation Officer—the DRO. This information will naturally be confidential and not for disclosure—except in so far as the patient may agree—to any member of the management. Health defects such as bad teeth or defective vision may be found to need early remedy; so there is an opportunity to see that curative treatment is provided. Reports to management following examination must be short and to the point; their value will depend on the doctor's degree of knowledge of the different occupations available. In the personnel or labour officer with understanding the medical officer has an ally whose value cannot be overestimated; he can well implement recommendations regarding shorter hours, for example, or instructions to the canteen manager concerning diet. Job allocation based on simple job analysis is not difficult and most men fit readily into broad groups. Much of the value of the first examination is wasted if there is no follow-up system, and in this respect the help of a good nurse is essential. In large firms the doctor could well have a trained social worker on his staff. Follow-up is obviously more important in cases where there has been doubt as to physical and mental stamina; many ex-prisoners of war, for example, will need special supervision for some time to come. Follow-up must include interview and, if necessary, re-examination; progress reports should be obtained from the man's manager, foreman, or supervisor; the man should be studied at work; and it is important to find out his reactions to the job, to his mates, and to 'the firm.' If these things are well done the doctor in industry can contribute much to that part of the problem which deals with resettlement in employment; by doing so he can add to the prestige of industrial medicine in no uncertain manner.

DUST AND LUNG DISEASE

In 1923 Collis drew attention to the fact that the iron oxide inhaled by haematite iron ore miners caused shadows in the skiagrams of their chests.[1] Since then McLaughlin and others have described the changes occurring in electric-arc welders and silver polishers.[2] [3] It is curious, however, that the only necropsy report on these lungs continues to be that by Enzer and Sander [4] who showed that while the perivascular and peribronchial lymphatic spaces were packed with iron oxide dust there was no fibrosis in the lungs. Prendergrass and Leopold [5] have recently suggested that this form of dust disease should be called 'Benign Pneumokoniosis,' and while this is a good term in contrasting it with other forms of dust disease, it must be strongly stressed that the lung cannot become a physiological dust trap and yet retain its normal structure and function. It would seem probable that heroic quantities of any dust, no matter how innocuous they might be in small concentrations, would in time overwhelm the defences of the lung and accumulate in such amount as to impair function. Such a form of lung disease would obviously be due to causes of a mechanical nature—the physical presence of large amounts of inert foreign material. There is a tendency to disregard this group of diseases since any disability is slow to develop. But just as a hundred years ago the necessity for drains was discussed and now is taken for granted, it is to be hoped that within a hundred years from now the fact that ventilation can prevent these diseases will be taken for granted too, and that they will fade into medical history as a thing of the past.

Not all dusts, however, are benign. Some, such as arsenic, may give rise to carcinoma; others, such as cotton (byssinosis) and platinum may give rise to asthmatic conditions. Still others, such as manganese, beryllium, osmium and bagasse may

1 *Proc. roy. Soc. Med.*, 1923, **16**, 85.
2 *Lancet*, 1936, **1**, 171.
3 *Ibid.*, 1945, **1**, 337.
4 *J. industr. Hyg.*, 1938, **20**, 333.
5 *J. Amer. med. Ass.*, 1945, **127**, 701.

give rise to acute inflammatory conditions in the lungs or bronchioles, while chronic inflammation may arise from exposure to the dusts of hay ('farmer's lung'), bagasse and vanadium, among many other substances. Nevertheless, far the most important dust in industry is that resulting from silica, and a great deal of work is still needed to explain the exact means by which changes in the lung caused by this substance are brought about. The report of experimental studies on chronic pulmonary disease in South Wales coalminers by King, Belt and Nagelschmidt recently published by the Medical Research Council,[1] and reviewed on another page, throws considerable light on the problem. They uphold the solubility theory of silicosis first propounded in 1932 by Kettle [2] when he found that the coating of a previously harmful quartz dust with an impervious layer of iron oxide rendered it quite innocuous when introduced into the lungs of guinea-pigs; and they discard the idea suggested by Peacock in 1860 [3] and Greenhorn in 1865 [4] that the fibrous reaction was scar tissue resulting from the grains of sand and their abrasive action on the soft alveolar and lymphatic tissues of the lung. Doubt was first thrown on this latter theory in 1923, when Gardner [5] published the results of his classic experiment with a dust of finely divided carborundum (silicon carbide) which failed to produce silicosis despite the sharp-edged nature of its particles.

Belt and King [1] show that when silica dust is introduced into the lung, it is first engulfed in phagocytes, and so long as these cells remain intact there is no reaction in the surrounding lung tissue. The liberation of small amounts of silicic acid within the cell probably tends to preserve the cell membranes intact and fibrosis will not result, unless an acute inflammatory reaction supervenes, either from fortuitous causes or as a result of silicic acid escaping from the koniophages. If this happens an extensive acute inflammatory process results which goes on to carnification which organizes with the heavy nodular reticular fibrosis of silicosis. This theory places major importance upon the relative solubility of the silica, and as King [1] has shown the apparent solubility of silica is depressed by certain substances, notably by aluminium, iron shale and certain silicates, though certain other silicates, among which asbestos is outstanding, seem to have fibrogenic powers themselves. Belt and King summarized by saying that the tissue reaction of silica is not always a simple matter of impact of silica on living cells but entails an interplay of adjuvant and antidotal factors; some of these, like the adjuvant effect of tubercle in human silicosis and the antidotal effect of aluminium, are known, while others, especially in experimental pneumokoniosis, are unknown and are not amenable to rigid control in animal dusting experiments.

The most surprising finding, however, is that while all the South Wales dusts have low silica solubilities, anthracite coals depress the silica solubility of quartz markedly more than do bituminous and steam coals, yet the incidence of severe pneumokoniosis is greater in anthracite miners than in the bituminous or steam coalminers. The low solubility of the South Wales dusts in general would imply a low toxicity, yet many of them are pathogenic and the anthracite ones highly toxic. King suggests that the low solubilities may be related to the release, from other components of the dust, of aluminium, which may form a protective coating area over the free silica particles. Within the alveolar spaces, and in the lymph tissues, the releasable aluminium may, however, eventually disappear from the sandstone dusts and from the particles of free silica, leaving them finally exposed to the inter- and intra-cellular fluid. Thus there may be produced in the lung mineral material from which all the releasable aluminium has been leached, leaving it potentially more pathogenic than it was at the time of its inhalation. Mineral dusts containing higher proportions of aluminous components should be more inert, even after long residence in the lungs, and this seems to be the case, since it is recognized that the shales are less dangerous to health than the sandstones.

While ideally the way to prevent one dust disease is not to expose the worker to another, these theories support the Canadian evidence that aluminium dust will prevent silicosis. Denny, Robson and Irwin [6] [7] showed that metallic aluminium dust of particle size below 5μ produced no fibrosis in rabbits' lungs and protected the animals against silicosis. On the other hand, Belt and King [8] repeated the experiment in rats and found that the lungs of these animals treated the aluminium powder as a foreign body and formed small concretions with fibrous tissue around the particles. However, Hunter, Milton and Perry [9] found no harmful effects from aluminium dust in grinders of aeroplane propellers, and Crombie, Blaisdell and MacPherson [10] investigated 125 workers employed in the Pittsburgh stamp mills of the Aluminium Company of America with similar results. Nevertheless, it is difficult to conceive that alumina would not produce the same changes and mechanical effects as iron oxide, even though it is less radio-opaque, but as a result of these findings

Crombie and his colleagues treated 34 silicotic miners by the daily inhalation of fine aluminium powder freshly ground from small aluminium pellets in a specially constructed mill. Daily treatment began with five-minute inhalations, which were gradually increased to 30 minutes. Some of the men received 300 treatments, but the majority only 200. Out of 34 cases thus treated, clinical improvement in 19 was manifested by lessening or disappearance of shortness of breath, cough, pain in the chest and fatigue. In 15 cases the condition became stationary, and remained so in spite of continuous employment in silica dust throughout the treatment. The progress of the disease was assessed by means of tests of respiratory function, repeated at three-monthly intervals.

This treatment cannot be regarded as a cure for silicosis, since it cannot restore lung tissue which has undergone fibrotic change, but obviously until the engineers are able to suppress dusts completely it must be used in increasing trials in attempts to prevent the onset of this disease. It is to be hoped, therefore, that such trials will not be impeded by the fact that the Canadians have patented the process.

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1 *Spec. Rep. Ser. Med. Res. Counc.*, 1945, No. 250.
2 *J. Path. Bact.*, 1932, **35**, 395.
3 *Trans. path. Soc., Lond.*, 1860, **12**, 36.
4 *Ibid.*, 1865, **16**, 59.
5 *Amer. Rev. Tuberc.*, 1923, **7**, 344.
6 *Canad. med. Ass. J.*, 1939, **40**, 213.
7 *Industr. Med.*, 1939, **8**, 133.
8 *J. Path. Bact.*, 1943, **55**, 69.
9 *Brit. J. industr. med.*, 1944, **1**, 159.
10 *Canad. med. Ass. J.*, 1944, **50**, 318.

IDEAL SEEING CONDITIONS *

THE STUDY OF THE HUMAN VISUAL SYSTEM AS A BASIS FOR PRESCRIBING LIGHTING

BY

J. H. NELSON

From The Joseph Lucas Research Laboratories, Birmingham

In the Illuminating Engineering Society's pamphlet on the lighting of factories, entitled 'Making Work Lighter,' [1] it is stated: 'Every-day experience is the only proof we need that much better lighting is necessary for seeing very small than very large objects, and scientific investigation has taught us just how much better it needs to be.' The pamphlet goes on to describe the scope of good lighting, pointing out that if the best seeing conditions are to be achieved care must be taken in the choice not only of the lighting installation, but also of the surfaces on which the light falls. Good industrial seeing conditions, in fact, result from a well-designed lighting installation in a tastefully decorated shop and with work suitably arranged. If such good seeing conditions are to be introduced into industry it is essential to have a clear picture of the ideals to be aimed at and how these ideals are deduced from the characteristics of the eyes. The study of the visual system must be carried out with the object of determining its performance in quantitate terms under defined conditions. Moreover, if a complete understanding is ever to be possible, the function of the visual system in terms of the physical concepts, such as visual acuity and brightness difference threshold, must be related to the anatomical details, such as the retinal neurons with their complex cross connections and differing modes of response to stimulation.

To build up a picture of the visual system it is essential firstly to study the simple performance of one eye. The thresholds such as visual and vernier acuity can be studied and related to the anatomical structure of the retina under certain idealized conditions. The responses of single nerve fibres and the electrical stimulation of the optic nerve can be studied for some lower animals and the results correlated with, for example, the intensity-time response of the human eye. Moreover these experiments indicate the importance of time as a factor in the study of visual reactions.

The more complete picture of the visual system with two eyes working interdependently as part of the brain is much more difficult to visualize. This is inevitable as our knowledge of the enormously complex control mechanism is rudimentary, as is our knowledge of the method of analysis of the nervous messages and their subsequent synthesis into a picture of the outside world. This difficulty must not be allowed to divert too much attention from the visual system as a whole to the more easily understood physical picture of monocular vision. The most complex of all factors to be taken into account in the study of the visual system is the effect of surroundings on the mood and behaviour of the observer. Some places or rooms are gloomy, others pleasing. The conditions leading to a sensation of cheerfulness must be known and defined even if the means by which they achieve their effect cannot be explained.

The simple study of the responses of one eye alone, the more complex behaviour of the two eyes giving a picture of the whole outside world, and finally the psychological effects of this picture must be studied and made a basis of an ideal set of seeing conditions. Realization in practice may or may not be possible, but ideals must not be bound by practicability. On the other hand, of course, recommendations in every real case must always be thoroughly practicable.

Monocular Vision

If an object is to be distinguished from its background it must satisfy four essential requirements:

(*a*) *Size*.—It must subtend a sufficient angle at the eye.
(*b*) *Contrast*.—The object must contrast sufficiently with its background.
(*c*) *Brightness*.—The brightness of the object must be sufficient to excite response.
(*d*) *Time*.—The object must be presented to the eye for sufficient time.

These four variables are interdependent and have been studied exhaustively by Luckiesh [2] for the special case of the visual acuity threshold. As a test object Luckiesh used two parallel black bars on a white background made by eliminating the middle third of a black square. This object corresponds closely to the Snellen type but is, in his opinion, superior.

The results shown in fig. 1 bring out clearly the importance of contrast and brightness in enabling the eyes to discriminate fine details. The importance of time is not shown very clearly though it is evident that the performance of the eye is superior for exposure times of 0·300 seconds to that for exposures of 0·075 seconds, which correspond approximately to the longest and shortest fixation pauses. Important as these results are, they do not give a complete picture of visual resolution. Three additional phenomena must be studied, namely (*a*) the effect of the surround field brightness on the discrimination for a constant test object brightness; (*b*) the existence of different types of resolution of which 'Vernier acuity' is the most important; and (*c*) the effect of

* A paper read to the Birmingham Group of the Association of Industrial Medical Officers on May 11, 1945.

[1] Lighting Reconstruction Pamphlet No. 6 (1945). Illum. Eng. Soc., Lond.

[2] Luckiesh, M., and Moss, F. K. (1937). *Science of Seeing*. Macmillan, Lond.

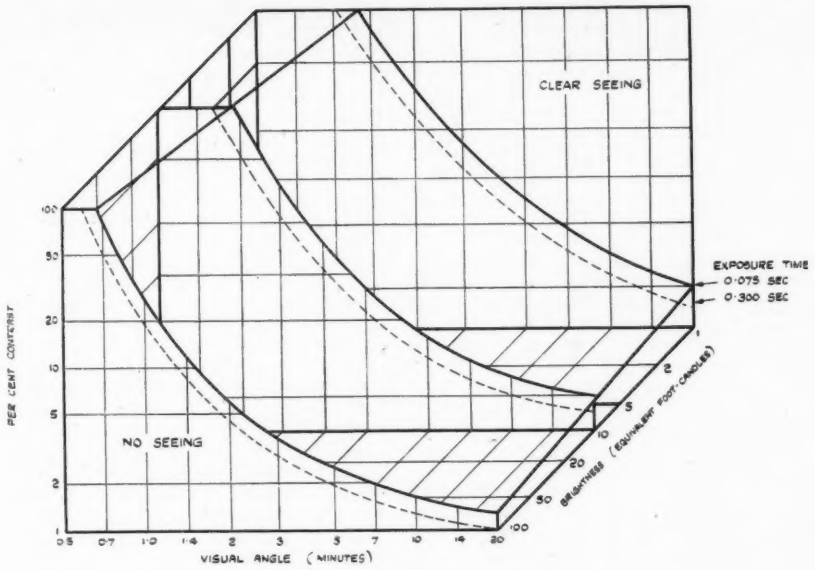


FIG. 1.—The threshold conditions. (After Luckiesh.)

supra-threshold conditions as opposed to the thresholds which are easily studied.

Lythgoe [3] studied the change in visual acuity with brightness under three sets of conditions:

(*a*) with a dark surround;
(*b*) with a surround of fixed brightness; and
(*c*) with the surround brightness equal to that of the best object.

As shown in fig. 2, these results are clearly of great importance and extend the threshold data given by Luckiesh,

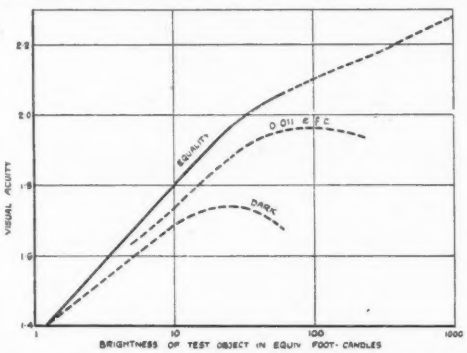


FIG. 2.—Visual acuity and the influence of the surround field. (Lythgoe.)

who has also performed exhaustive experiments on the effect of the surround field. These experiments show conclusively that the performance of retinal element or a small group of elements cannot be studied in complete isolation but only in relation to other retinal elements in its neighbourhood or more strictly as a part of the whole visual system.

The phenomena of contour or Vernier acuity is further evidence of the beneficial operation of these retinal cross-connexions. It is found that if part of a straight line in the field of view is displaced then a displacement subtending an angle of 10 seconds of arc at the eye can be detected by a normal observer, a much finer degree of resolution than the 1 minute of arc which is taken as normal for the Snellen type test for visual acuity.

Before considering the relation between the two types of visual resolution and the structure of the retina it is worth while considering the scope of Vernier acuity in every-day life. In all probability it is more important than visual acuity and it is somewhat unfortunate that it has not been more extensively studied. Fig. 3 attempts to formalize the types of visual resolution and shows how vernier acuity and spacial perception are related, as has recently been described by Walls.[4] In addition, we shall later see that Vernier acuity and stereoacuity are related. Since the retina is built up of a very large number of discrete photo-receptors the 'grain-size' of the retina must set a limit to the possible discriminating power of the eye. In terms of visual acuity, if we are to discern the white space between two small rectangles, then the image of the white space must fall on one retinal element while the two black bars fall on the cones each side. More exactly one cone must be stimulated less than its two neighbours by an amount corresponding to the brightness difference threshold for a single cone. In the fovea centralis, that part of the retina devoted to the seeing of fine detail, we find only cone elements, each of which has usually a one to one connexion through the retinal neurons to the optic nerve and the minimum of cross-connexions. This is in contrast to the rods several of which join to one optic nerve fibre, thus helping their greater sensitivity to light, which is essential as they are concerned with night vision. The foveal cones are, moreover, found to be very densely packed. Their average diameter is 2–3 microns (or 0·001 inch), so that if the image of the white space of the visual acuity test object is 6 microns wide there will be one cone completely unstimulated. If the minimum perceptible

[3] Lythgoe, R. J. (1932). *Spec. Rep. Ser. Med. Res. Counc.*, No. 173.

[4] Walls, G. L. (1943). *J. opt. Soc. Amer.*, 33, 487–505.

retinal image is taken as 0·0002 inch then at a distance of 10 inches from the eye a detail of 0·003 inch can be discerned, and this corresponds to a limiting angle of about 1 minute of arc, which is gratifying agreement with experiment.

A more exhaustive examination of the nature of the retinal image shows that real conditions are far more complex than the simple geometrical image formation pictured above. However it appears that while the image of even the smallest object falls on several cones a very small difference in stimulation is sufficient for discrimination.

A quantitative relation between the retinal structure and Vernier acuity is not so straightforward. With retinal elements subtending about 30 seconds of arc one can attain, at the best, a discrimination of 2 seconds of arc. The mechanism by which this is achieved is somewhat similar to that enabling one to see a clear picture at the cinema, where each individual picture is of very poor quality. The act of discrimination depends upon a large number of retinal elements working together, and since they are arranged in random fashion it is possible to position a line more exactly than the width of a single element.

A further problem in discrimination arises, namely, the finest black line that can be detected on a white background (fig. 3). This again seems to be a case in which a number of elements work in conjunction, and it is found that a line subtending as small an angle as 0·5 second of arc can be seen under favourable circumstances. The simplest explanation of this phenomenon seems to be to consider it as a case of brightness difference and calculate the percentage area of a cone shielded by the geometrical image of the line. This works out at about 1 per cent., which compares favourably with the best performance for brightness difference threshold, for a large number of cone elements working together.

So far there has been little indication as to why the performance of the eye depends so very largely on the brightness of the visual field. It is probable that the clue lies in the time required for nervous response, and the experiments described so far do not bring out the importance of this factor. Luckiesh has adopted the argument that since the unconscious eye movements limit the eye's rest pauses to between 0·075 second and 0·30 second, we need not consider time intervals beyond this range. This argument may be valid when we are considering practical application of good lighting principles, but there is ample evidence that in the study of nervous responses we must consider a greater range of time intervals.

Two lines of investigation are being followed which

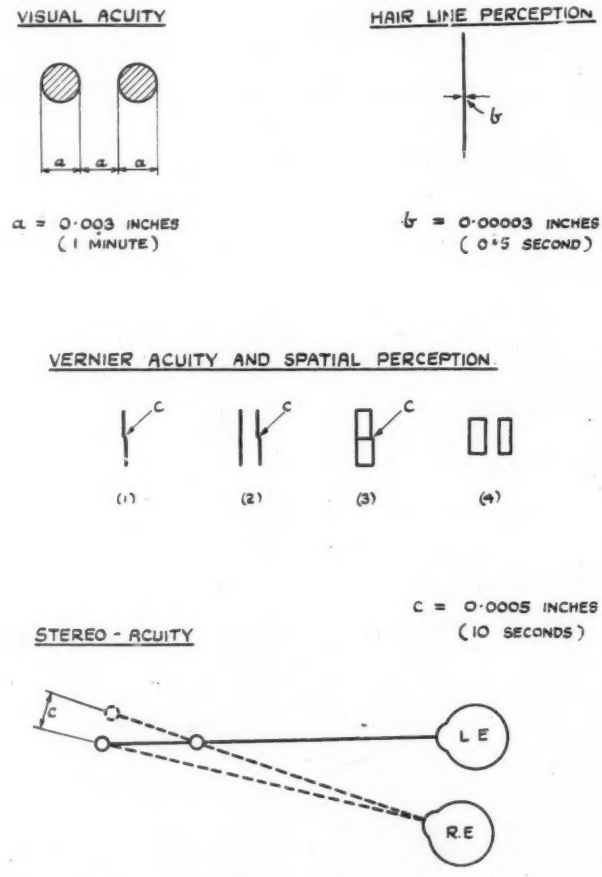


FIG. 3.—Types of visual resolution.

should eventually lead to a better understanding of the nature of the retinal response. Polyak [5] has made an exhaustive study of the retinal elements and their cross-connexions in the inner layers of the retina. As a result of this work he has listed a number of different types of cone connexion, indicating possible differences in function. This is in general agreement with the findings of Granit [6] and Hartline,[7] who have by different techniques analysed the electrical response of a bundle of fibres and of single nerve fibres respectively. The experiments of Hartline illustrated in fig. 4 indicate the effect on the response of changes in both the intensity and duration of the stimulus. It can be seen that over a very wide range not only the strength of response but also the latent period between stimulus and response depends upon the energy of the stimulus. Remembering that the eye movements effectively limit us to those responses which have commenced within 0·3 second, the influence of intensity on the power of discrimination becomes obvious.[8]

These experiments on the nature of the nervous responses have been carried out on the eyes of lower animals. In so far as they show the fundamental nature of these responses the results can be applied to the formulation of a pictorial theory of vision, but if we are to have a quantitative theory it is evident that exact information on the behaviour of the more complex human retinal elements must be available.

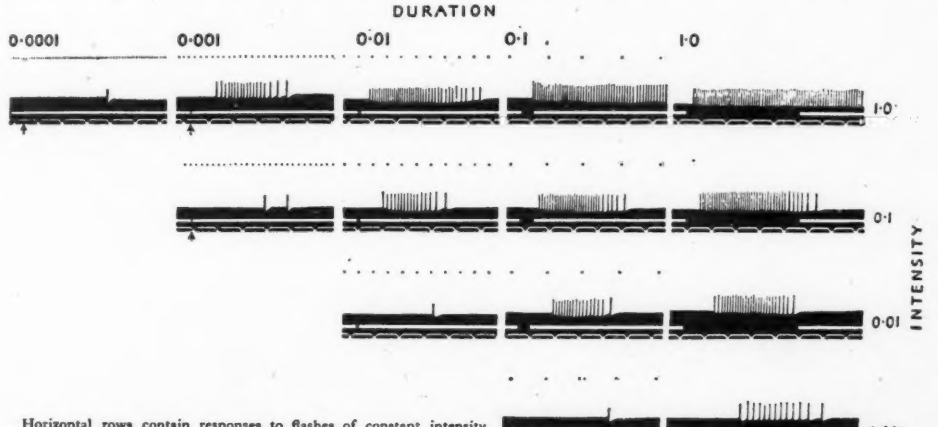
Binocular Vision

The study of the fundamental thresholds, visual acuity, contour acuity and the brightness difference threshold, and their relations with the structure of the eye, is necessarily a study of monocular vision. However, under ordinary working conditions we are concerned with binocular vision, and it is essential to relate our studies to the visual system as a whole. The connexions between the two eyes and the brain, of which the retina by its structure must be considered an outpost, are well known. The optic nerves of the two eyes pass to the chiasma, where they are divided so that fibres from the right-hand side of each retina pass to the motor sensory region of the right lobe of the brain, and those from the left-hand side to the left lobe. It is probable that the nervous messages are partially analysed at this stage. For instance, it is thought that anatomical evidence of a triple mechanism to account for colour vision has been found. In particular what might be called the synchronizing messages must be filtered out at this stage and control impulses sent back along the efferent paths to bring about the exact co-ordination of the eyes. From the motor sensory regions the visual radiation transfers the modified nerve messages to the occipital cortex where the final sensation of sight takes place.

Polyak [5] has traced the course of the principal nervous messages from their stimulation by the image of the outside world through the chiasma to the occipital cortex. His diagram brings out well how the small central field surrounding the fovea is given pride of place and occupies a very large space in the ' picture ' received in the cortex. The study of stereoacuity, which may be defined as the smallest angular difference between the two pictures that can be detected, shows that stereoacuity is of the same order as Vernier acuity. Fig. 3 brings out this relation in the idealized case of two vertical rods of different height, one of which is in front of the other. The left eye sees the far rod exactly behind the front one, but the right eye sees the nearer rod to the left-hand side of the far one by about 10 seconds of arc for the limiting condition. The image of the right eye has a difference corresponding to the Vernier acuity limit between its image and that of the left eye. This difference is, however, interpreted as one of position in depth.

There seems to be little quantitative data on the relation between the field brightness and stereoscopic resolution. This is unfortunate as the brightness of the picture is very important, as can be demonstrated by comparing

[5] Polyak, S. L. (1940). *The Retina.* Chicago University Press.
[6] Granit, R. (1933). *J. Physiol.*, 77, 207.
[7] Hartline, H. K. (1934). *J. cell. comp. Physiol.*, 5, 229.
[8] Wright, W. D. (1938). *The Perception of Light.* Blackie, Lond.



Horizontal rows contain responses to flashes of constant intensity and varying duration. Duration of flash is given in seconds.
Vertical columns contain responses to flashes of constant duration and varying intensity. Intensity is given in arbitrary units.
Responses on a diagonal correspond to a constant product of (duration × intensity).
The notched white lines record the time of the response in fifths of second. Above the notched line is a white line on which is recorded the duration of the light flash. For very short flashes, its position is shown by an arrow.

FIG. 4.—Response from single optic nerve of Limulus for various intensities and duration of stimulation. (Hartline.)
(With acknowledgments to the Journal of Cellular and Comparative Physiology, U.S.A.)

a stereo-print with a stereo-transparency. The latter, having a higher brightness and a great range of contrast, gives a far more realistic appearance.

Psychological Effects of Background and Colour

In studying the threshold conditions for seeing details, it has been evident from the work of Lythgoe and, later, Luckiesh that the power of discrimination depends not only upon the conditions of the details under examination but also upon the surroundings. This is so, not only in the narrow sense that the immediate surround field affects the ability to discriminate detail, but also in the wider sense that the brightness of the surroundings have a profound influence on the behaviour and response of the observer. In a general way the artist has for centuries been aware of the effects which colour harmonies can have, and the great masters have used this knowledge in the composition of their pictures. A few of the ancients have attempted to formalize the effects of colour patterns on the observer, and some have even made attempts to lay down the relations which must exist between the constituent colours of a pattern. Until recently, however, this work has been limited by the fact that the range of coloured pigments available for experiment was very restricted, and also there existed no rational method of specifying colour. In recent years the number of pigments and dyes has been increased so that the range of colours now available is very wide. A matter of greater importance is that the Munsell system of colour specification has been put on a sound basis, though it may not yet fulfil all the requirements that the most exacting might ask.

The early work of Newton and later Young has made the idea of a colour triangle familiar, being based on the experimental fact that all known colours can be specified as mixtures of three suitably chosen primaries. Practical considerations have also led to the specification of a colour as a mixture of a given spectral colour with so much white. The Munsell colour tree (fig. 5) may be thought of as a development of this latter. The greys, from black to white form the trunk, while colours of the same 'hue' are arranged on radial planes so that there is a progressive increase in the 'chroma' or strength of the colours as their distance from the central trunk increases. This colour tree has two great advantages for practical application, (*a*) it is simple and orderly, and (*b*) the scales are chosen so that a Munsell step in any direction corresponds to the constant change in sensation.

Colour Harmonies

In the study of the relationship, first described by Moon and Spencer,[9] between two colours which has been restricted to the special case of the eye being adapted to a Munsell colour N5 the following possibilities are found:

(*a*) Identity.
(*b*) First ambiguity.
(*c*) Similarity.
(*d*) Second ambiguity.
(*e*) Contrast.
(*f*) Glare (limited to 'value').

[9] Moon, P., and Spencer, D. E. (1944). *J. opt. Soc. Amer.*, 34, 46, 93, 234.

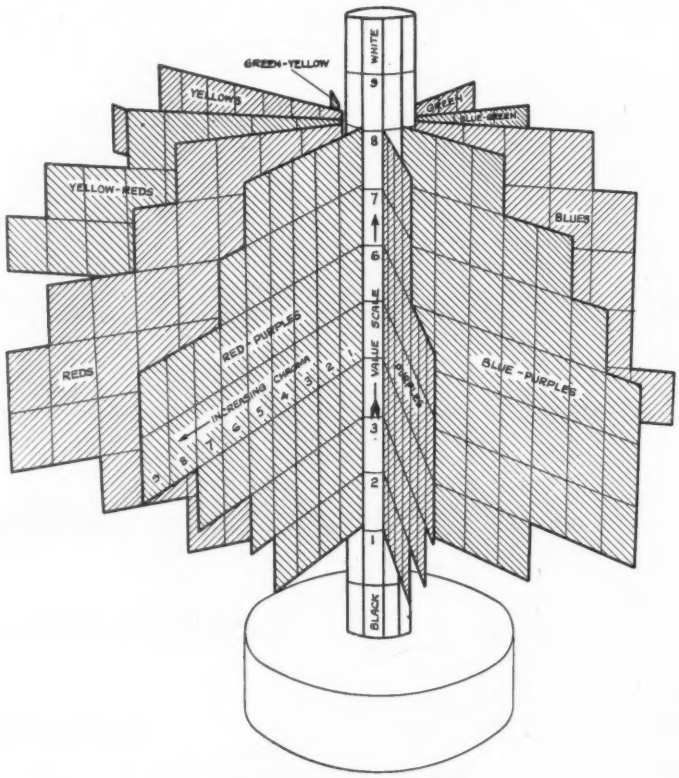
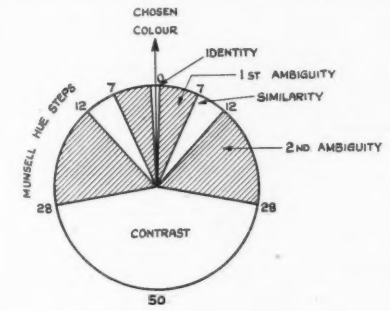


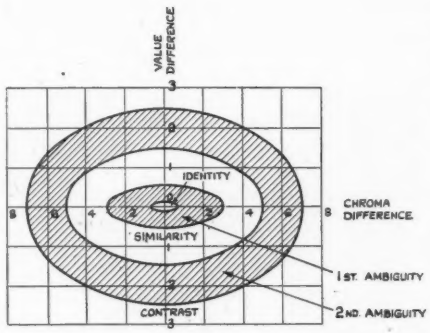
FIG. 5.—The Munsell colour tree.

Of these it was evident that when the relationship was definite the effect was on the whole pleasing, but when the relationship was doubtful, as when two colours were so nearly the same that one was not sure if they were intended to be the same, then the effect was displeasing. In addition, of course, an excessive difference in 'value' was unpleasant as it produced glare.

In fig. 6 the regions corresponding to the five principal relations between the colours are shown for two cases, (*a*) a plane of constant value and for colours of any chroma, (*b*) a plane of constant hue for colours varying



(*a*) REGIONS OF SIMILARITY AND CONTRAST IN A PLANE OF CONSTANT "VALUE."



(*b*) REGIONS OF SIMILARITY AND CONTRAST IN A PLANE OF CONSTANT "HUE."

FIG. 6.—Regions of similarity and contrast in the Munsell colour tree.

in both value and chroma. One of the two colours is supposed to be at the point 0.0. in the second case and the other may be anywhere in the pattern. Since all Munsell steps correspond to nearly the same sensation interval the relations illustrated can be imagined to be anywhere in the colour solid.

Harmonies of a number of Colours

When more than two colours are to form a harmony, it is found that their points in the Munsell colour solid should form some regular geometrical figure. For example, a pleasing harmony of three colours of the same value would form a triangle, four colours a square, and so on. Such geometrical figures are, of course, not limited to the simple case of colours of constant value but may equally well lie in a plane of constant hue of even an oblique plane through the colour solid.

Area Balance

It is well known that when one selects a sample of wallpaper the small cutting produces a completely different effect from the large expanse on the wall. The effect on a dark colour is apparently to increase in darkness as the area is increased and similarly a very light colour will increase in lightness. These effects have been known and used by artists for centuries, but such an effect can only be described with any degree of exactness in terms of some measurement.

It will be remembered that the colour harmony experiments were carried out with the eye adapted to a neutral tint of value 5, and under these circumstances it was found that if two coloured cards of the same size were to have equal importance the colours must lie at the same distance from the point N.5. in the colour solid. Supposing, however, the two colours were at different distances from the adapting colour then the ratio of their areas must be inversely as their distances from N.5. More exactly it is found that the moment of one colour about the point N.5. must either equal or be some multiple of that of the other colour.

Psychological Effects of a Colour Scheme

The rules of colour harmony state in simple geometrical terms what colours or groups of colours may be used together and in what quantities, but give no indication of the effect the pattern will have on the beholder nor how it will compare with any other pattern obeying the same rules. It is found that the reaction of an observer to a particular colour harmony depends on two factors: (*a*) the average brightness of value of the whole pattern, and (*b*) the mean colour, which would be produced by mixing all the colours composing the pattern in proportion to their areas. These two factors are, of course, interdependent in their action, the significance of the mean colour being negligible when the value of the pattern is low, but increasing and becoming dominant for a high value. Table 1, which is taken from Moon and Spencer's original work, indicates the range of psychological effects to be expected.

TABLE 1

PSYCHOLOGICAL EFFECT OF BALANCE POINT

Munsell Hue	Munsell Value	Munsell Chroma	Psychological effect
R	—	>5	Very stimulating, very warm.
YR	—	>5	Stimulating, warm.
Y	—	>5	Slightly stimulating, slightly warm.
GY	—	>5	Slightly restful, neutral temperature effect.
G	—	>5	Restful, slightly cool.
BG	—	>5	Very restful, very cool.
B	—	>5	Non-stimulating, cool.
PB	—	>5	Non-stimulating, cold.
P	—	>5	Slightly stimulating, neutral temperature effect.
RP	—	>5	Stimulating, slightly warm.
Any	>6·5	Any	Cheerful.
Any	<3·5	Any	Gloomy.
Any	Any	<5	Non-stimulating, neutral temperature effect.

From these results it can be seen that if the mean value of a pattern is below 3·5 (10 per cent. reflectivity) it will always appear gloomy to an observer adapted to a mean value of 5 (20 per cent. reflectivity), but a pattern of mean value above 6·5 (37 per cent. reflectivity) will appear cheerful. Further, it is only for patterns having a value greater than 6·5 that the effects of the mean hue and chroma become significant. Of these the patterns of low chroma (i.e. less than chroma 5) have no specific effect, but the high chroma patterns have definite psychological effects depending on the dominant hue.

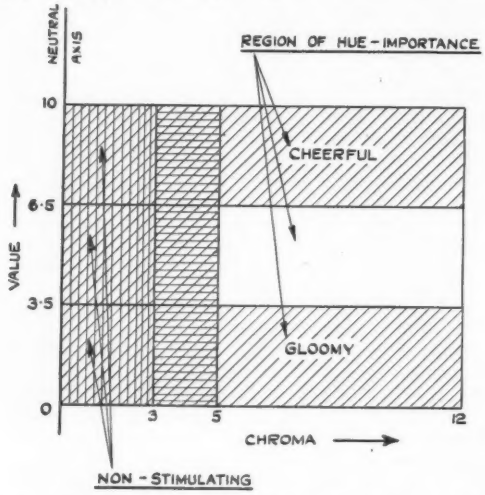


FIG. 7.—Illustrating the regions of the Munsell colour tree giving a specific psychological effect.

These results are shown graphically in fig. 7, which represents a plane of constant hue in the Munsell colour solid, and can be considered as typical of all hue planes.

Ideal Seeing Conditions

To specify the best possible seeing conditions experiments should be carried out to determine the nature of the operation of the eyes under conditions considerably above the threshold. Such experiments are extremely difficult to perform as there is no obvious method of measuring the ease of seeing. Luckiesh [10] has used the involuntary blinking test as a means of indicating the readability of type. Other workers disagree with this method, but do not seriously challenge his conclusions. More recently Weston [11] has attempted to formulate a rational basis for prescribing illumination to give at least a 90 per cent. efficiency. The illumination required is based on that used to yield a given visual acuity and a nomograph can be of great practical help when used in conjunction with a thorough job analysis, a problem that often proves very difficult.

Interpreting the lessons from the study of threshold seeing and from the study of the psychological effects of the surroundings the following are suggested as a basis for ideals.

(*a*) Details should be as large as practicable—e.g. about 1/100 inch.

(*b*) Critical contrasts should be as high as possible without causing glare—say 100 to 1.

(*c*) The central field brightness should be of the order of 1000 equivalent foot-candles.

(*d*) The immediate surround field should be comparable in brightness with the central field, which with the more remote surroundings should be so coloured to yield variety without distraction.

(*e*) The eyes of the operator should either be relaxed or accommodated well within the comfort range for the individual.

[10] Luckiesh, M., and Moss, F. K. (1942). *Reading as a Visual Task*. Chapman and Hall, Lond.
[11] Weston, H. C. (1943). *Trans. illum. eng. Soc.*, Lond., **8**, 17.

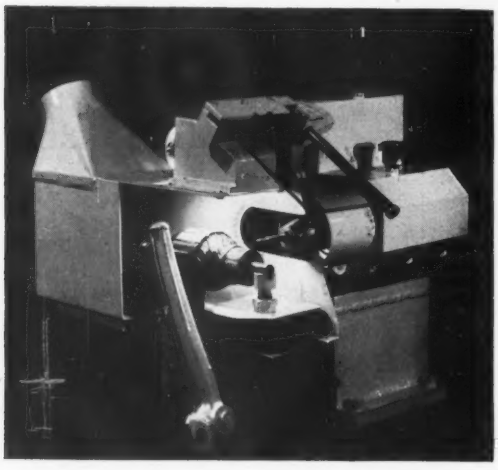


FIG. 8.—Commutator under-cutting machine.

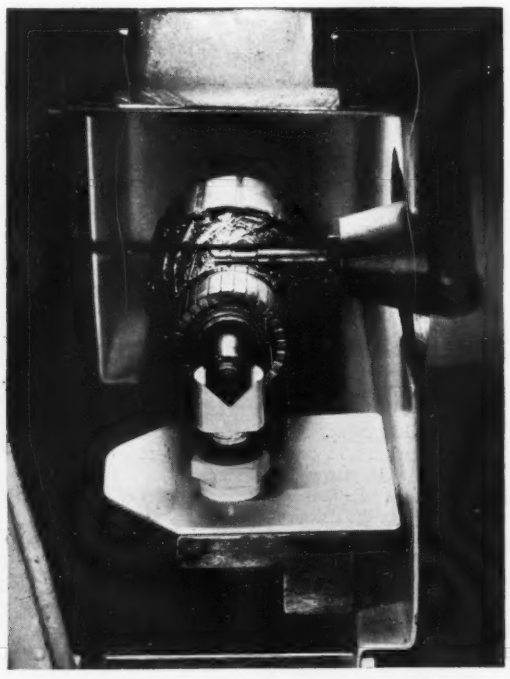


FIG. 9.—Commutator under-cutting machine (view of work).
(*By courtesy of Sheet Metal Industries.*)

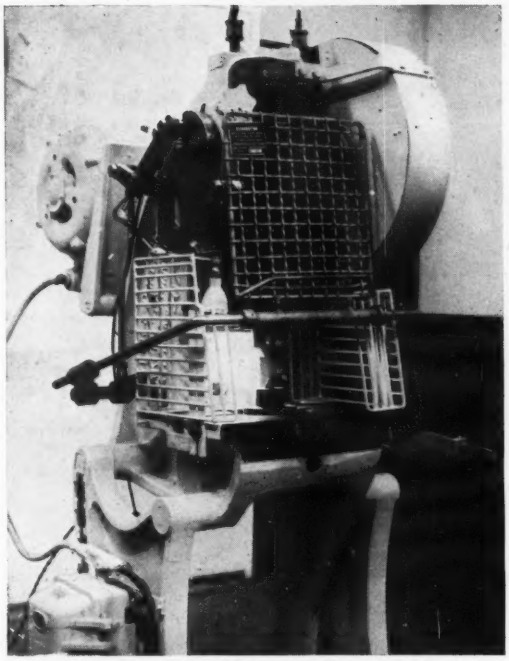


FIG. 10.—Small press with local lighting and finished in a light colour.

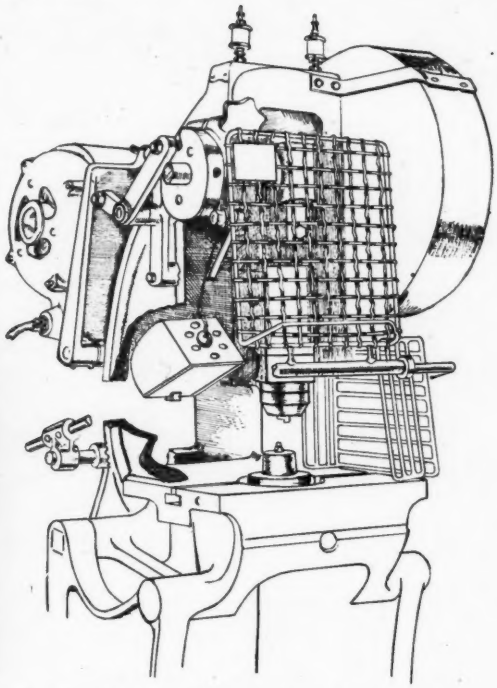


FIG. 11.—Cut-away diagram of press showing lighting unit in position.

(By courtesy of Sheet Metal Industries.)

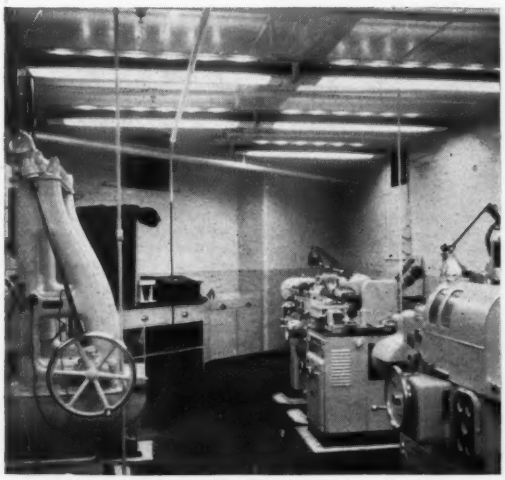


FIG. 12.—' Jig-boring ' room with light dado.

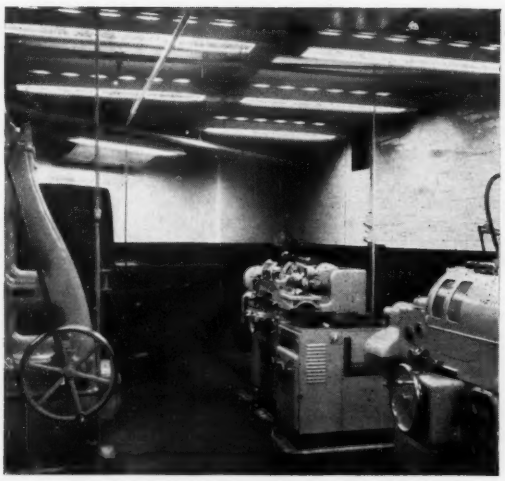


FIG. 13.—' Jig-boring ' room with dark dado.

(By courtesy of Sheet Metal Industries.)

FIG 14.—General view of tool room.
(*By courtesy of Sheet Metal Industries.*)

Practical Applications

It is, of course, not always possible to achieve these ideals in practice, both for technical and economic reasons. There are, however, many cases where seeing conditions can, with advantage to both worker and management, be made very good indeed. A typical example of such a case is illustrated in figs. 8 and 9, which show a small special purpose machine designed for undercutting the mica between the copper segments of a small commutator.[12] An initial brightness of 1500 equivalent foot-candles was obtained by using an illuminated opal panel, a reflection of which was seen in the work. By means of suitably arranged surrounds, having brightnesses in the region of 50–150 equivalent foot-candles, this high work brightness was made comfortable.

The opal panel is a very powerful tool for the lighting of metal surfaces and a further example of its use on a small power press is illustrated in figs. 10 and 11.[13] The cutaway diagram in fig. 11 shows how the lighting unit is fitted between the side frames in such a way that it gives maximum efficiency with a minimum of interference.

Decoration

Control of the remote surroundings of the visual field cannot be effected with the exactness with which the central visual field can, in many cases, be prescribed. Particularly in an industrial interior, the pattern presented to the eyes is an extremely complex one. The choice of a suitable decoration must depend very largely on the particular circumstances and should have for one of its objects the suppression of distracting elements in the field of view.

In table 2 the details of two industrial decoration schemes are given. That shown in fig. 12 is suitable for a small shop, where the walls form an important proportion of the field of view. The use of a dark dado, so common in many industrial interiors, is not to be recommended for the small space, fig. 13, but is suitable for a larger shop as shown in fig. 14. The two schemes illustrated are applicable to the light engineering industry, and whilst the same principles will apply to all industries their application must be altered to suit the peculiarities of the industry concerned.

[12] Nelson, J. H. (1944). *Sheet Metal Industries*, Jan. and Feb.
[13] Nelson, J. H. (1945). *Ibid.*, Apr.

TABLE 2

SAMPLE INDUSTRIAL DECORATION SCHEMES

Location	Scheme shown in fig. 12.	Scheme shown in figs. 13 and 14.
Ceiling	White (reflectivity 75 per cent.)	White.
Walls	Broken White (reflectivity 60 per cent.).	Duck Egg Tint (reflectivity 60 per cent.).
Dado	Eau de Nil (reflectivity 45 per cent.).	Holly Green.
Machine Tools	Portland Stone (reflectivity 55 per cent.).	Portland Stone.

Summary and Conclusions

The importance of good seeing conditions is becoming recognized in the more progressive sections of industry, and there is a genuine desire to translate this recognition into practice.

If the best results are to be obtained the study of the eyes from the point of view of the physiologist, physicist and psychologist must be directed towards the specification of an ideal set of conditions. The present state of our knowledge of vision is examined in this paper from this point of view.

The relation between the performance of the eye and its structure is shown in the case of visual acuity, and the significance of the time response of the retinal elements in explaining the dependence of the performance on brightness level is pointed out.

The psychological effects of colour and the lightness of the surroundings are described and related to the practical problem of decoration. Examples of this are given by illustrations of machine tools and industrial interiors.

REVIEWS

CHRONIC PULMONARY DISEASE IN SOUTH WALES COALMINERS. III.—EXPERIMENTAL STUDIES

Medical Research Council, Special Report Series, No. 250

(H.M. Stationery Office, London. 1945. Pp. 94. 5*s*.)

Nos. 243 (1942) and 244 (1943) of the Special Report Series were devoted to the study of chronic pulmonary disease in South Wales coalminers; the first was a clinical and pathological study, and the second an environmental study. The present report is a sequel to these and is concerned with experimental studies. It is the greatest contribution to our understanding of silicosis yet made. Broadly there are three sections: the first a study of the mineral content of the lungs of some 54 miners by King and Nagelschmidt, the second an investigation into the tissue reactions in the lungs of rats caused by selected dusts from the South Wales coalmines by Belt and King, and the third a study of the solubility of the dusts by King.

The lungs of the 54 workers in the coal-field were analysed chemically for silica, alumina, and coal; and the quartz, mica (sericite) and kaolin were determined by x-ray diffraction-pattern analysis in the minerals contained in the lungs which were isolated as residues. The results of these studies showed that the composition of the dust in the lung reflected the nature of the man's employment, and was identical with the dust to which he had been exposed at work. The pathological lesions in the lungs showed no relation with either the coal or kaolin in the dust; but the concentrations of mica and quartz ran more or less parallel with the degree of fibrosis observed.

Pure or 'clean' coal, that is mineral that contains more than 90 per cent. coal, whether it is anthracite, bituminous or steam coal, produced in animals nothing more than the simplest type of foreign body reaction with mineral fibrosis. There was some concomitant emphysema and decreased vital capacity, and the animals lived six months less than controls. The histological picture was similar to the dust reticulation of coalminers. On the other hand, coal dusts with a siliceous content of more than 12½ per cent. produced a reticulation fibrosis, a type of organization similar to carnification. Mica and kaolin were often present and the amount of quartz was small, thus there is evidence that some silicates may be fibrogenic, though on the other hand the silicates comprising clod strata (shale) appeared to inhibit the fibrogenic properties of quartz. Specimens of pure mica (sericite) from South Wales produced an absolute minimum of reticular fibrosis; whereas a sample of mica (hydromuscovite or sericite) from another source used as a control, produced heavy nodular reticulin fibrosis similar to quartz.

The incidence of acute inflammatory reaction to a dust was closely parallel with the higher grades of fibrogenesis. The initial impact of silica is on the phagocyte, and it is not until this is disintegrated that it falls on the connective tissue. The most severe reactions undoubtedly result from quartz, but the pathogenicity of this substance is reduced by clean coal and practically abolished by shale. The result is dependent evidently on an interplay of adjuvant and antidotal factors, the adjuvant effect of tubercle in human silicosis and the antidotal effect of aluminium are known, but there are probably many others which still remain unknown.

There is little doubt that the production of silicosis depends on the solubility of silica. It is proved that quartz particles coated with iron-oxide or alumina are less soluble and less harmful than those which are not. It is surprising, therefore, to find that all South Wales dusts show low silica solubilities. Shales depressed the solubility apparently by releasing aluminium from their aluminous components to form a protective covering which could be stained with aurine, over the quartz particles; this effect, however, was not related to the total aluminium content of the sample. One of the most perplexing findings, however, is that anthracite coals depressed the silica solubility of quartz markedly, whereas steam and bituminous coals did so very much less; yet severe pneumokoniosis has a much higher incidence in anthracite workers. King suggests as an explanation that in some way bituminous and steam coal inhibit the solubility of the siliceous matter contained therein, whereas anthracite has not this property of combination (the content of siliceous matter is the same in all three), and so its mineral content can produce its effect, either under experimental conditions by inhibiting the solution of added quartz, or under conditions in the mine by not preventing the solution of siliceous material as other coals do. K. M. A. P.

LE TRAITEMENT DES ACCIDENTS DU TRAVAIL MANUEL DU PRACTICIEN

By J. Boudreaux, A. Hanuat and M. Iselin (with the help of Ch. Duvelleroy)

(Foundation Francaise pour l'etude des Problemes Humains. Librairie de Medicis, Paris. 1945. Pp. 163. Fr. 81)

This work gives an account of the treatment of minor industrial accidents which are defined as those which can be dealt with satisfactorily without the help of general anaesthesia. The ground covered is therefore fairly wide, although not everyone would agree that conditions such as fracture of the scaphoid and distal end of the radius should be treated in this manner. The book is primarily intended for the French general practitioner-surgeon who undertakes minor traumatic surgery, and the introduction gives in some detail the organization of a service of this kind not only describing the layout of the buildings required but also indicating the duties of the nursing staff as well as giving a note on the necessary materials and instruments. Stress is rightly laid on the devotion of adequate time by the operator to the procedures described and it appears that the ill-results which follow work done in haste by poorly trained surgeons in indifferently equipped surroundings are to be met with in France as well as on this side of the Channel.

The main part of the book is, like Gaul, divided into three parts which deal respectively with what are called 'closed injuries' (contusions, sprains, dislocations, fractures and lumbago of traumatic origin), 'wounds,' and 'specialties.' Under the last named are chapters by appropriate specialists on ophthalmology, oto-rhino-laryngology and stomatology. There is a short appendix on the emergency treatment of accidents.

The text is written in clear simple language and in general follows modern practice, but it is an indication of the scientific isolation in which France has found herself until recently that a book published in 1945 should have no mention of the use of penicillin. Noteworthy also is the absence of reference to the rehabilitation of the injured workman and the important part played by the industrial medical officer in this phase of treatment. Our French colleagues seem to have something to learn from us about this. The book has a good table of contents but lacks an index. G. K.

MEMORANDUM ON CARBON MONOXIDE POISONING

Factory Dept., Ministry of Labour and National Service

(H.M. Stationery Office, London. 1945. Pp. 32. 6*d*.)

So much has been written in the technical and physiological literature on the effects of carbon monoxide on man and animals, on blood in vitro and in vivo, and on tissue respiration that it would almost seem superfluous to issue further information on the subject. But it will be apparent to all who have to do with industrial acci-

dents that one can neither maintain too high a pressure in the drive to prevent accidents nor can one exhaust the infinitely various list of possibilities of industrial mishap. This memorandum, recently issued by the Factory Department, is no brilliant exposition, but is a solid and sensible attempt to warn people of the commoner ways in which such poisoning may occur.

Statistics of accident and fatality due to CO for the years 1941–2–3 are given; these show that the annual incidence is fairly steady between 225 and 240, while deaths undergo some variation between 5 and 10 per cent. of the incidence. It must be remembered that these cases refer to those which caused more than 3 days disablement and so reflect probably only a small fraction of those who have experienced symptoms from CO. It would be of considerable interest to know at what levels of saturation workers find it necessary to stay off work for 3 or more days. It is significant that such noted investigators as Henderson and Haggard state emphatically that a claim for compensation should be rejected unless the claimant is exposed for at least 3 hours to conditions that could or did lead to 50 per cent. saturation of the blood, and was completely unconscious for at least 6 hours after return to fresh air. 'Short of conditions of such severity,' they state, 'recovery is practically always complete.' Such criteria mean, of course, that there is likely to result from such exposure irreversible lesions, probably in the central nervous system and brought about by the anoxaemia.

It seems generally agreed nowadays that the conception of chronic CO poisoning is fallacious. The memorandum under review states: 'Carbon monoxide is not intrinsically a poison; it exercises its asphyxiant action not through any inherent toxicity in the carboxyhaemoglobin formed but simply through this power of using up most of the haemoglobin, which is the transporter of oxygen to the tissues.' As a physiological description of the action of CO this is very well but we suggest that it is unwise to talk of CO as 'not a poison' especially to a public not composed mainly of physiologists and in view of the sentence on the first page of the memorandum where it states: 'poisoning by CO is the cause of between one-half and one-third of all reportable gassing accidents which occur in factories.'

It must be confessed that the industrial circumstances in which CO poisoning occurs are difficult to overcome especially where hand operations are in use, for example in manual charging of blast furnaces. Dependence on wind direction and ample space to avoid escape of gas seems poor comfort these days; the fact is that many of our plants are out of date and it is time we set our house or factories in order. The memorandum offers useful hints on how to minimize the hazards in various operations and instances safety rules drawn up by a steel-making firm for the instruction of workers. This is a valuable inclusion in a Government pamphlet. Instruction of workers in the dangers inherent in this work is not as universal as it ought to be. The various arguments adduced against this would all become irrelevant if intelligent co-operation were obtained between workers, their unions and employers.

In the description of the effects of various concentrations of CO the memorandum closely follows the work of Henderson and Haggard, but departs from them in recommending subcutaneous strychnine or pituitary extract as of great value in serious cases. These authorities, with whom we agree, condemn all hypodermic, intravenous and intracardiac medications as not only valueless but even harmful in the field. The great thing in treatment of a serious case is to re-establish respiration at the earliest moment by the Schäefer method, assure an adequate intake of O_2 and CO_2, and combat shock. The memorandum rightly stresses that the first few seconds may determine everything and save life.

In the matter of methods of detection and estimation of carbon monoxide in the atmosphere, it could have been helpful if a short critical review could have been included of the various methods recommended. For our part the only reasonable method is a continuous recorder, as otherwise the usual sequence will be calamity followed by determination of concentrations.

M. W. G.

OUTLINES OF PHYSICAL METHODS IN MEDICINE

By G. D. Kersley, M.D., F.R.C.P.

(William Heinemann, London. 1945. Pp. 85. 6*s*.)

Physical medicine is a curious and rather illogical division of medicine, and when it goes beyond the normal group of treatment, massage, electro-, helio- and hydrotherapy, and includes occupational therapy, it is more illogical still. The author is right when he says that the abysmal ignorance and consequential lack of interest of many of the medical profession in physical medicine invited exploitation by the charlatan and provided little encouragement to those legitimately and conscientiously applying these matters. Doubtless the concept of rehabilitation will right these wrongs. No physician or surgeon can regard the treatment of his patient as complete until that patient is satisfactorily re-employed and this will undoubtedly require new methods. It would be better that the orthopaedic surgeon and the specialist in rheumatic diseases each controlled his own treatment; this is so in other countries. However, if the division of physical medicine is persisted with in England, then the closest liaison must be established with other branches of medicine. This little book, which gives an elementary account of the methods of physical medicine, is greatly to be welcomed at the present time and will enable the medical student and practitioner to acquire a knowledge of the subject.

K. M. A. P.

A GUIDE TO HEATING, VENTILATION AND LIGHTING

By W. Douglas Seymour

(Oxford University Press, London, New York and Toronto. 1944. Pp. 90. 6*s*.)

The author of this short book sets out to explain heating and ventilation in terms which will be understood by 'ordinary people, who by profession are probably baker, banker or billiard-marker.' But he does not fully succeed in his purpose, because, unconsciously, his language rapidly becomes technical, for example where he discusses methods of measuring temperature or the significance of daylight lighting. However, the potential misfortune of the billiard-marker is undoubtedly the fortune of the industrial medical officer. The book is at once short, clear, comprehensive, and inexpensive, and is one therefore that can be confidently commended to doctors employed in industry, not as a volume for permanent reference but as an effective introduction to a complex subject. The book is divided into two main parts: heating and ventilation, and lighting. The effect of these phenomena on our ability to work is explained in simple terms. If conditions are made comfortable at the place of work—the ultimate aim of heating, ventilation, and lighting—then productive effort is not interfered with, so there is mutual benefit to all parties. The author asks himself the question 'Why are heating and ventilation necessary?" and gives the answer in simple physiological terms. This leads to chapters on old and new ideas; measurements of temperature; room comfort; and some description of modern methods of heating and ventilating. Similarly, the answer to the question 'Why do we need light?" supplies the material for the second half of the book. Vision is possible only where there is light, so here is an immediate relationship between medicine and engineering. This link for the industrial medical officer is one of paramount importance; the more he knows about the material working environment the better can he apply his clinical knowledge. It is no doubt selfish to suggest that the book would have been improved, from our point of view, by more adequate references and by the provision of an index.

A. E.

ABSTRACTS

The Disabled Ex-Serviceman. PEP Broadsheet No. 238 (1945). PEP, 16 Queen Anne's Gate, S.W.1. Pp. 20.

Among the returning men and women we shall feel a special sense of obligation to those whose war service has caused lasting disability. They will have to overcome serious physical and psychological handicaps as part of the process of adjustment to ordinary civilian life. The job is made easier than after the last war: first, because great strides have been made in surgery and therapeutics, i.e. in the field of rehabilitation; second, by the corresponding progress in the field of resettlement, including wide-awake legislation of the Disabled Persons (Employment) Act, 1944. This Act provides for industrial rehabilitation courses of 6–8 weeks to ' tone up ' disabled workers. (The first centre for this has been opened by the Ministry of Labour at Egham.) It also provides vocational training courses of 6–12 months at Government Training Centres, and at special centres (such as St. Loyes College, Exeter, and Queen Elizabeth's Training College, Leatherhead) for the seriously handicapped. There is also to be a Register of the Disabled; compulsory employment of a quota of registered disabled; and the formation of a non-dividend-paying company to provide sheltered employment. A National Advisory Council is already in operation to advise the Minister of Labour on these matters, and District Advisory Committees are appointed to advise locally.

Rehabilitation, training, placement in suitable jobs, and, in certain cases welfare follow-up, are therefore all within the scope of the Government scheme. While this is good in theory, in practice something is going wrong. Placement in the right job is becoming difficult; there should be better welfare follow-up; there may be inadequate co-operation between hospitals and the Ministry of Labour. The Disablement Rehabilitation Officer—the DRO—is generally criticized; it is alleged that they are insufficiently trained, have inadequate knowledge of industrial work, have little understanding of the man's background, a too casual approach, and have not the right type of education or experience for the work. The field of recruitment should therefore be widened. Selected ex-servicemen, after a certain amount of training, would be specially acceptable to men from the Forces. Welfare and labour officers of the Ministry of Supply, now seeking other employment, might be suitable. There is need for better co-ordination of the national scheme, need for publicity, and the responsibility of industry itself must be more readily accepted by employers.

Memorandum on Dermatitis. (1944). LEEDS JOINT COUNCIL ON INDUSTRIAL MEDICINE.

Dermatitis is a general term indicating inflammation of the skin which may result from external or internal causes. Industrial dermatitis is the term applied when such inflammation results from contact with particular dusts or liquids capable of causing damage to the skin and encountered in the course of industrial occupations. Similar dermatitis is caused by other dusts and liquids encountered outside industry as in household duties. It may arise because of the irritant properties of the dust or liquid, from inadequate protection or from inadequate cleansing after work. It may also arise from idiosyncrasy or from sensitiveness of the skin to a dust or liquid normally harmless. Such personal predisposition can to some extent be determined by medical examination before employment.

Industrial dermatitis is responsible for much loss of man-hours, but it is, in the main, avoidable. It is not a disease which can be passed from one individual to another, is not contagious or infectious, and there is no reason why it should not clear completely if appropriate measures are taken. Industrial dermatitis is the reaction of a sensitive skin to some substance of which it is intolerant. The potential irritant properties of substances naturally vary. It is essential to appreciate that if the skin of a particular individual is provoked to a rash by a particular substance with which he makes contact in his work then as a rule he will recover if removed from that contact or if he is effectively protected from contact. Return to contact with the offending substance will cause a return of the rash unless effective protection is afforded and should not be permitted without medical sanction.

To avoid industrial dermatitis it is of prime importance to have a clean industry and a clean worker. Unnecessary contact between the worker and the dusts or liquids with which he works should be avoided. Dust extractors, ventilators, splash guards, washing facilities and provision of suitable ' cleansers ' are important. Thought should be given to the method of drying the skin after washing, e.g. by hot air dryers and automatic individual towels. Clothing should protect as much as possible of the worker from dusts and liquids, and should resist and throw off dust and liquid and not absorb them or bring them into more intimate contact with the skin. In some cases a change of clothing is necessary, e.g. in powder factories or mines. The skin that must be exposed may be effectively protected by the use of appropriate barrier creams applied to the skin of exposed parts before starting work. These leave on the skin an invisible and imperceptible glove which remains intact until the end of work when washing will remove the cream with the dust or liquid. Appropriate barrier creams are made to protect against almost all industrial hazards. If barrier creams are not employed the use of appropriate cleansers after work may be indicated, e.g. sulphonated castor oil with 2 per cent. wetting agent.

Attention is drawn to the danger of using such things as paraffin, turpentine, thinners, suds, abrasives, strong caustics, for cleaning the skin, or using any cleansers other than those recommended for the particular process.

Where these measures are employed care should be taken to receive and convey exact instructions concerning their use as they may be ineffective when improperly applied. It must be obvious that some individuals by reason of their sensitive skins or for other reasons are unsuited to certain trades and in such cases no protective measures will render them suitable.

Publications:*

Memoranda:

- Prevention of Dermatitis. Factory Form 1863.
- Prevention of Oil Rashes. Factory Form 296.
- Dermatitis among French Polishers. Factory Form 1797.
- Dermatitis in the Boot and Shoe Trade (Stock Rooms). Factory Form 1814.
- Prevention of Industrial Dermatitis, with special reference to the use of Barrier Substances. Factory Form 330.
- Prevention of Industrial Dermatitis: Dermatitis from Glues used in Aircraft Construction. Factory Form 331.

Cautionary Notices:

- Dermatitis. Factory Form 367. 2*d.* (3*d.*)
- Oil Dermatitis. Factory Form 397. 2*d.* (3*d.*)
- Dermatitis: Caution to workers handling flour and dough or sugar. Factory Form 355. 2*d.* (3*d.*)
- Effects of Lemon and Orange Peeling on the Skin. Factory Form 396. 1*d.* (2*d.*)
- Effects of Chrome on the Skin. Factory Form 398. 1*d.* (2*d.*)
- Dermatitis from Glues (Synthetic). Factory Form 366. 1*d.* (2*d.*)

* Memoranda obtainable from the Factory Department, Ministry of Labour, St. James's Square, London, S.W.1. The Cautionary Notices may be purchased from H.M. Stationery Office (prices in brackets include postage).

Bacterial Flora of Wounds and Septic Lesions of the Hand. WILLIAMS, R. E. O., and MILES, A. A. (1945). *J. Path. Bact.*, **57**, 27.

This paper reports a bacteriological survey (mainly in terms of aerobic bacteria) of 1197 wounds and septic lesions of the hand, the majority resulting from industrial accidents. The bacteriological technique is described and discussed in some detail: samples were inoculated on to 2 blood agar plates, for aerobic and anaerobic incubation, and into cooked meat medium which was subcultured on to blood agar after 2 days' incubation. Addition of the anaerobic plate and the cooked meat culture led to a notable increase in the number of swabs from which pyogenic cocci were isolated. Over 90 per cent. of the cases of sepsis could be accounted for in terms of infection with *Staph. aureus* and *Str. pyogenes*; it was also noteworthy that fresh wounds, swabbed within 6 hours of infliction, and healing wounds that never showed signs of sepsis frequently yielded the same organisms: 21 per cent. of 432 fresh wounds showed contamination with *Staph. aureus*, and 37 per cent. of 278 apparently healthy healing cuts were similarly infected. There was some evidence that the staphylococci found in fresh wounds represented, in large part, self-infection from the skin of the patient. Among septic wounds and other septic lesions, *Staph. aureus* was the predominant pathogen; and the majority of infections that develop as closed lesions, e.g. pulp space abscesses, and which presumably arise from microbes introduced into the tissues at the time of injury, were due to *Staph. aureus*. Those that develop as open lesions, e.g. paronychias and large septic lacerations, commonly yielded *Str. pyogenes* as well. It is suggested that the majority of wound infections are due, in the first place, to *Staph. aureus* and that *Str. pyogenes* is commonly added later to wounds already infected.

Work in the Heat as affected by Intake of Water, Salt and Glucose. PITTS, G. C., JOHNSON, R. E., and CONSOLAZIO, F. C. (1944). *Amer. J. Physiol.*, **142**, 253.

The best performance of fully acclimatized young men on a good daily diet, performing intermittent hard work in the heat, is achieved by replacing hour by hour the water lost in sweat. Any amount of water less than this leads in a matter of hours to serious inefficiency and eventually to exhaustion. Six men were exposed to a dry heat of 100° F. 30 per cent. relative humidity and a moist heat 95° F. 83 per cent. relative humidity. They marched up grades at 3·5 miles per hour for six hours a day with a ten-minute break at the end of each hour. Without water the rectal temperature rose to 103° and the men felt exhausted; if they drank at the same rate as they lost sweat the temperature remained steady and they did not feel tired. Normally men at work never voluntarily drink as much water as they sweat. The ill effects of progressive depletion of the salt reserves are well known, but these men had adequate salt in their diet. The serum chloride remained remarkably constant during prolonged work in heat. Replacement of the salt lost in the sweat hour by hour had no demonstrable advantage. With small doses of glucose (25 g. in 30 ml. water) there was a small but definite advantage over experiments when no water was permitted. When 25 g. of glucose hourly were administered in addition to water equal to the sweat, no significant advantage in favour of glucose could be detected with the possible exception of a slightly slower pulse rate. No advantage was detected from administering 200 mg. of ascorbic acid, 20 mg. thiamine hydrochloride, 20 mg. of riboflavin or large doses of brewer's yeast, either singly or together, either during work or the day before. When practical problems of transportation and supply, lack of appreciation of the importance of water and salt or the anorexia which is so common in hot environments, interfere with adequate intake, it may become desirable to supply salt in the drinking water, or less satisfactorily in the form of tablets.

Physiologically Equivalent Conditions of Air Temperature and Humidity. ROBINSON, S., TURRELL, E. S., and GERKING, S. D. (1945). *Amer. J. Physiol.*, **143**, 21.

Experiments were made to determine the most severe environmental conditions in which men could maintain thermal equilibrium after the second hour of six-hour exposures. The experiments were made in men wearing shorts or army jungle uniforms in air-conditioned rooms with dry-bulb temperature readings between 23° C. and 50° C. with various relative humidities. An air movement of 55 m. per minute prevailed during the experiments. Experiments were made with the man (*a*) sitting, (*b*) walking at easy pace on the treadmill, (*c*) performing moderate exercise in the treadmill. Observations were made on the elevation of the heart rate, rectal temperature, skin temperature and rate of sweating. Men walking in shorts with metabolic rates of 188 Cal./m.^{2} per hour maintained thermal equilibrium for six hours at 34° C. with 91 per cent. humidity and at 50° C. with 21 per cent. relative humidity. With metabolic rates of 130 Cal./m.^{2} per hour they maintained equilibrium at 35° C. with 96 per cent. humidity and at 50° C. with 32 per cent. humidity. With resting metabolic rates of 46 Cal./m.^{2} per hour men in shorts could maintain equilibrium at 36° C. with 98 per cent. humidity and at 50° C. with 34 per cent. humidity.

Bibliography of Industrial Hygiene 1900–1943. BELLINGHAM, ELLEN F., BLOOMFIELD, J. J., DREESSEN, W. C. (1945). *U.S. Publ. Hlth. Bull.* No. 289. Washington. Pp. 95. 20 cents.

This bibliography has been prepared in response to demands from the medical profession, industry, and labour in the U.S.A., 'for a list of material on industrial hygiene.' The authors state that it does not attempt to be a complete list (nevertheless it is comprehensive and will be helpful to those engaged in industrial medical research).

Iron Oxide Dust and the Lungs of Silver Finishers. MCLAUGHLIN, A. I. G., GROUT, J. L. A., BARRIE, H. J., and HARDING, H. E. (1945). *Lancet*, **1**, 337.

Silver polishers inhale iron oxide from the rouge used at their work. Three workers were examined clinically and radiologically and a fourth who died, following an operation for gastric ulcer, had a necropsy. In each case the skiagrams of the chest were comparable with that found in electric arc welders. The necropsy showed bulky, heavy lungs with bullous emphysema. There was pneumonic consolidation of both lower lobes. Microscopical examination showed extensive acute broncho-pneumonic and well marked emphysema. There was no evidence of chronic inflammation and there was no fibrosis. Three kinds of pigment were present: that enclosed in phagocytic cells found in aggregates under the pleura, and along the pulmonary vessels, this was probably iron oxide; a fine granular pigmentation of the elastic laminae of the pulmonary arteries and of the elastic fibrils in the alveolar walls, this was silver; and lastly iron pigment stainable with ferrocyanide present in small amounts in intra-alveolar phagocytes. Chemical examination of the lung showed that ash constituted 10·17 per cent. of the dry weight, and of this ash 72 per cent. was iron (reckoned as Fe_2O_3) and 6 per cent. was silver (as metal). The x-ray changes were considered therefore to be mostly due to iron oxide but in part to silver. X-ray pictures of normal sponges resemble a lung somewhat. They were soaked in graded suspensions of iron oxide (rouge), ranging from 5 to 60 grains in a pint of water. The excess fluid was squeezed out and they were again radiographed. Stippled shadows appeared in the films of these rouge-containing sponges, varying in density with the concentrations of iron oxide particles. This proved that iron oxide will cause these shadows in the x-ray. Little or no physical disability appeared to be caused to the men by the presence of the iron oxide dust in the lungs, though in one case there was emphysema. A worker

who has inhaled radio-opaque dusts may therefore have an x-ray picture of his lungs simulating silicosis or miliary tuberculosis, and yet have no obvious physical disability.

Benign Pneumonoconiosis. PENDERGRASS, E. P., and LEOPOLD, S. S. (1945). *J. Amer. med. Ass.*, **127**, 701.

Eleven grinders were examined. The metal ground in the manufacture of bearings is chrome vanadium and chrome molybdenum tool steel containing about 98 per cent. iron, 2 per cent. alloy and not more than 0·2 per cent. silica. Artificial abrasive wheels composed of bakelite, silicon carbide (carborundum) and aluminium oxide (aloxite), have been exclusively used. The dust concentration averaged 4,800,000 particles per cubic foot of air, of which 96·5 per cent. were less than 5μ and 99·5 per cent. less than 10μ. The average percentage of silica as quartz in the dust was 0·43 per cent. Four of the workers who had been grinders for 12, 13, 14 and 17 years showed x-ray changes of nodulation. One man showed a coincident pulmonary tuberculosis in the x-ray, though he had no clinical manifestations of active infection. It was considered that these were 4 cases of siderosis occurring in metal grinders.

Observations on Pulmonary Disease in Graphite Workers. DUNNER, L. (1945). *Brit. J. Radiol.*, **18**, 33.

Inhaled pure graphite dust can produce pneumokoniosis. Five cases are reported. The symptoms and signs may be strikingly mild, even in advanced cases. Case 1, aged 56, had worked with graphite for 20 years. He had cough, sputum and pain in the chest. Skiagram showed patchy infiltration throughout the right lung. Case 2, aged 63, had worked with graphite for 17 years. He had cough and sputum, several specimens of which contained no tubercle bacilli. Skiagram showed a massive reniform opacity in the right upper zone with a similar but less well marked opacity in the left. Case 3, aged 58, had worked with graphite for 20 years. He had no symptoms, but a skiagram showed reticulation in both lungs. Case 4, aged 57, had worked with graphite for 34 years. He had been 'chesty' for 10 years. Skiagram showed peripheral fibrosis in the right upper zone, with some nodulation and increased linear striation. Case 5, aged 42, had worked with graphite for 18 years. He had no complaints, but a skiagram showed rounded opacities in both infraclavicular regions.

Clinical Physiological Observations on Welders with Pulmonary Siderosis and Foundry Men with Nodular Uncomplicated Silicosis. ENZER, N., SIMONSON, E., and EVANS, A. M. (1945). *J. industr. Hyg.*, **27**, 147.

The existence of siderosis, or the deposition of iron in the lung, has recently been discovered in arc welders, though the occupation has not hitherto been considered to cause disablement. In order to investigate the question more thoroughly a group of 15 arc welders with siderosis was compared with eight patients suffering from silicosis and 8 to 100 normal subjects. A number of functions were investigated including endurance in dynamic work (lifting loads) and static work; muscle strength; pulse rate before and after exercise; fusion frequency of flicker during rest and after exercise; maximum tapping rate; vital capacity; and maximum pulmonary ventilation. No statistically significant difference between the normal group and the siderosis group was observed in any function, though the silicosis group showed a significant depression of vital capacity, maximum pulmonary ventilation and endurance in dynamic and static work. When, however, the extent of nodulation in the x-ray films was graded from 1+ to 4+, the siderosis subjects were found to show nodulation ranging from 1+ to 3+, and averaging 2·2+. When the complex working capacity of the individual subjects was compared by means of an arbitrary scoring system composed of the results of the most significant tests, the total score expressed the individual differences and group differences more significantly than any of the single functional tests. The average score of the siderosis workers was 1·5, whilst that of five silicotic foundrymen who were more than 50 per cent. disabled from manual labour was 33·4, and that of five foundrymen still at work was 4·2.

Memorandum on Welding Processes and their Hazards. (1945). LEEDS JOINT COUNCIL ON INDUSTRIAL MEDICINE.

1. *Forge Welding.* The blacksmith's principle of heat and pressure is used in certain modern electric welding processes such as spot welding, seam welding, projection welding, and butt and flash butt welding. In these no fluxes or slags are used and there is no added metal.

(*a*) *Spot Welding.* Used on fairly thin metal sheets. The overlapping edges of the metal are placed between two copper electrodes and pressed together. The electrodes themselves provide the necessary heat and pressure to effect a weld.

(*b*) *Seam Welding.* The overlapping sheets are pushed through a pair of disc electrodes which gives a continuous linear weld instead of a series of single joints as in spot welding.

(*c*) *Projection Welding.* The machine is in the form of a press the flat plates of which are the electrodes. One of the sheets to be welded has small projections made on its under surface which form points of contact with the lower sheet. As the electrodes are brought together under pressure a series of spot welds are formed at the projections while the sheets are being pressed together.

(*d*) *Butt and Flash Butt Welding.* Used for joining lengths of heavy sections of steel end to end. The two pieces are held in electrodes in the form of a vice and as the ends are brought together an arc is struck and welding takes place.

2. *Autogenous or Fusion Welding.* A local melting and pooling of the metals to be joined takes place with or without the addition of molten metal from a filler rod or electrode.

(*e*) *Gas Welding.* Oxy-acetylene—propane—hydrogen—butane—coal gas. Filler rods, non-coated, usually the same but may be of different composition from the metal being welded. A flux may be used with some metals.

(*f*) *Metal Arc Welding.* Heat supplied by arc struck between metals to be joined and an electrode. Both parent metal and electrode melt and fill in jointing space. Electrodes are usually designed to deposit similar metal to that of parent metal being joined, and are coated by means of dipping, wrapping or extruding or a combination of these methods. The purposes of the coating are: (1) Stabilization of the arc. (2) Production of a flux and of a protective slag to cover the hot metal during cooling. (3) To add alloying elements to the weld metal. (4) To provide a gas shield round the arc to prevent access of air to the weld metal.

Examples of electrode coatings: sodium silicate; iron oxides and silicates (e.g. haematite and kaolin); potassium aluminium silicates (felspar, mica, talc); blue asbestos, $NaFe(SiO_3)_2$; and fluorspar, CaF_2.

(*g*) *Carbon Arc Welding.* Carbon electrode carried in holder. Heat supplied by arc and, if added metal is required, filler rods must be used as in gas welding.

(*h*) *Atomic Hydrogen Welding.* Arc is struck between two electrodes carried in holder and a stream of hydrogen (or cracked ammonia) is blown into arc. This is in effect a special form of gas welding as the melting is due to the high temperature flame produced and not to the arc.

Hazards

Health risks are confined to the second group of autogenous or fusion welding processes.

(1) *Electric Shock.* Very small risk with D.C. at 50–100 volts. Greater danger with use of A.C. Factory Department recommend D.C. with voltage not exceeding 60. Nevertheless A.C. is being increasingly employed as for D.C. a rotary converter is required. Electric

shock at voltages employed may have severe effects though seldom fatal, but a shock to a welder may result in a fatal accident by causing a fall when working in dangerous positions as on scaffolding or in shipbuilding.

(2) *Explosion risk in Gas Welding.*

(3) *Radiation Effects.* (*a*) Infra-red—heat effects may give rise to cataract and burns. (*b*) Light radiation—painful effects on eyes. (*c*) U.V. radiation—serious effects on eyes. The welder himself seldom suffers as he is familiar with the risks. Much lost time is caused in industry by the effect of flash on those passing by or those working near welding operations.

(4) *Burns* from hot metal thrown off from arc during welding.

(5) *Eye injuries* from chipping off slag from weld after cooling.

(6) *Fumes.* (*a*) *Iron oxide* is known to be deposited in the lung and to give a characteristic x-ray appearance in films but is not known to cause fibrosis or other ill-effects. (*b*) *Manganese.* Cases of manganese poisoning have been reported when welding steels containing 12–14 per cent. manganese. (*c*) *Zinc, cadmium, nickel or chromium* have all been reported as causing metal fume fever. (*d*) *Lead* poisoning cases have occurred during welding of lead alloys, lead covered plates and lead painted plates. (*e*) *Silica and asbestos.* No danger so far as is known. (*f*) *Fluorides.* Unpleasant fumes but no toxic effects reported. (*g*) *Nitrous fumes.* Potentially dangerous. (*h*) *Ozone and carbon monoxide.* No toxic effects reported.

(7) *Heat effects and anoxaemia* in confined spaces.

Precautions

D.C. under 60 volts. Ventilation—general and localized exhaust. Protective clothing, aprons, gloves, face shields and goggles. Gloves not only protect from burns but are the welder's main protection from electric shock. These are usually of leather and are protective when dry but are conductors when wet. A better material than leather is required but has not yet been discovered.

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Memorandum on Electric Arc Welding—Form 329 (Third Edition). Factory Department, Ministry of Labour.

Memorandum on Safety Measures Required in the Use of Acetylene Gas and in Oxy-Acetylene Processes in Factories—Form 1704. Factory Department, Ministry of Labour.

Precautions in the Use of Welding and Cutting Gases—Admiralty Shipbuilding Memoranda. Sheet No. 77.

X-ray Appearances of the Lungs of Electric Arc Welders—Doig, A. T., and McLaughlin, A. I. G. (1936). *Lancet*, **1**, 771.

Carbon Monoxide: Its Hazards and the Mechanism of its Action. VON OETTINGEN, W. F. (1945.) *Publ. Hlth. Bull.* No. 290. Washington. Pp. 257. 35c.

CO poisoning is characterized by the formation of CO haemoglobin. The amount of CO haemoglobin formed depends on the concentration of CO in the air, the duration of the exposure, and the rate of respiration of the individual. Increase in temperature, humidity, and CO_2 content of the air, or decrease in O_2 concentration, stimulate respiration and favour absorption of CO. The toxic effects of CO resulting in anoxaemia are due also to the CO haemoglobin further reducing the O_2 supply by interfering with the dissociation of oxyhaemoglobin available. The formation of CO haemoglobin also results in a reduction of the available reduced haemoglobin and in this way interferes with the transportation and dissociation of CO_2. How far CO also reacts with other " heme "-containing pigments and enzymes needs further study. Concentrations of CO haemoglobin in the blood below 20 per cent. cause only moderate subjective complaints; these become serious with concentrations up to 30 per cent. and alarming with higher concentrations. Concentrations between 60 and 80 per cent. are dangerous to life. Analysis of the clinical picture and pathological findings indicates that the sequelae of CO poisoning are more serious than would be expected from mere anoxaemia of a similar degree; there may be a specific primary or secondary effect of CO on " heme "-containing pigments or enzymes of the organism. In treatment, a prompt elimination of CO from the organism is vital: this is best done by administration of O_2, accompanied, if necessary, by artificial respiration. The value of simultaneous administration of CO_2 is discussed and its potential dangers pointed out. The use of morphine and atropine is contra-indicated.

Polarographic Determination of Toxic Metal Fumes in Air. LEVINE, L. (1945). *J. industr. Hyg.*, **27**, 171.

This paper outlines the procedures necessary for the polarographic determination of atmospheric fume samples of lead, zinc oxide, cadmium, chromic acid and manganese. Substantially the same operations are performed for each of them, the method being a rapid one, suitable for routine determinations. Moreover, two or more toxic metals can be determined simultaneously. In general, about a cubic metre of the air is sampled by means of the modified Greenberg-Smith impinger or the electrostatic precipitator. The sample is treated with volatile acids and evaporated to dryness. The salt is taken up with a suitable volume of the proper supporting electrolyte solution, and the polarogram is recorded photographically. The concentration of fume is measured by comparing the polarogram with a calibration curve prepared by photographing a series of standards. The sensitivity of the polarographic method for estimating lead fumes is considerably greater than that of the chromate-iodometric method, though not as great as with the dithizone method. A number of technical details are given for sampling each of the fumes mentioned, but they are not suitable for abstracting. The principles of the polarograph have previously been discussed by several investigators.

A Rapid and Simple Method for Measuring Small Amounts of Cyanide Gas in Air. ROBBIE, W. A., and LEINFELDER, P. J. (1945). *J. industr. Hyg.*, **27**, 136.

By the method to be described a concentration of HCN as low as 0·2 gamma in 10 litres of air (1 part in 50,000,000) can be measured. It has been used repeatedly for over a year with consistently satisfactory results. A measured air sample is bubbled through a solution consisting of three parts of 0·005 M. $Na_2HPO_4.12H_2O$ and one part of reagent (made by adding 1 ml. of 0·5 per cent. phenolphthalin in absolute ethanol to 99 ml. of 0·01 per cent. $CuSO_4.5H_2O$ solution). One part of 0·1 per cent. KOH solution is added, and the red colour which develops is measured after one minute with a photoelectric colorimeter. The HCN concentration is then determined by means of a curve prepared from readings of standard KCN solutions. The phosphate buffer reagent eliminates the effect of the CO_2 in the air. Five tables of data are recorded showing the accuracy of the method under various conditions, and information is given about substances in the air which may interfere with the reaction.

Acute and Subacute Toxicity of Di (2-Ethylhexyl) Phthalate with Note upon its Metabolism. SHAFFER, C. B., CARPENTER, C. P., and SMYTH, H. F., Jr. (1945). *J. industr. Hyg.*, **47**, 130.

Di (2-ethylhexyl) phthalate has the trade name of ' Flexol ' Plasticizer DOP. It is widely used in the production of flexible films and tubing. It is a light-coloured liquid, insoluble in water, but miscible with most organic solvents. Single doses of it (in a 1 per cent. solution) were administered by stomach tube to rats and rabbits, and the animals were observed for 14 days following the dose. The lethal dose (for 50 per cent. of the animals) was calculated to be 30·6 g./kg. for rats and 33·9 g./kg. for rabbits. Single intraperitoneal injections of rats with undiluted phthalate gave a similar figure, and skin absorption by rabbits gave a result not significantly different from that recorded for the stomach.

Over a period of 90 days groups of five rats were given oral doses of the phthalate every day mixed with the diet. The dosage was 0·2–1·9 g./kg., and no rat died, though growth was somewhat retarded. Experiments were also made on two adult human subjects, who swallowed single doses of 10 g. and 5 g. respectively of the phthalate. The former case showed mild gastric disturbance, but the latter, no symptoms whatever. The ester is completely saponified in the body, and in both men *o*-phthalic acid equivalent to 4·5 per cent. of the dose was recovered from the urine. Dogs showed a similar recovery rate, but in rabbits it amounted to no less than 26–65 per cent. The investigation, taken as a whole, shows that di (2-ehtylhexyl) phthalate is a chemical of low toxicity, and that the health hazards involved in its use as a plasticizer are slight.

Carbon Disulphide and Hydrogen Sulphide: Clinical Study of Chronic Low-grade Exposures. RUBIN, H. H., and ARIEFF, A. J. (1945). *J. industr. Hyg.* **47,** 123.

It was stated by Barthelemy, as the result of ten years' experience in the manufacture of viscose rayon, that no morbidity was observed if the CS_2 air content was kept below 30 p.p.m. and the H_2S below 20 p.p.m. Other investigators reported psychic symptoms, especially extreme irritability and insomnia. The present investigation was made over a period of four years on 100 individuals, of whom 78 were on a three-shift system and the remainder on day shifts. Air samplings were made regularly, and they showed a range of 1·9–26·4 p.p.m. of CS_2 (mean, 12·2), and of 1·0–5·5 p.p.m. of H_2S (mean, 2·5). The subjective symptoms indicated that 32 per cent. of all the workers suffered from fatigue at the end of the day's work, and that 26 per cent. of the shift workers were tired on awakening, as contrasted with 9 per cent. of the day workers. An occasional blurring of vision was experienced by 2·6 per cent. of the shift workers, and impaired appetite by 5 per cent. of them. The tendon reflexes were increased in 23 per cent. of the entire group, whilst corneal reflexes and hearing were in each case diminished in 16 per cent. of them. The investigators concluded that the symptomatology is vague and difficult to evaluate, and that there was not any sign or group of signs indicative of chronic intoxication.

A Study of Perchlorethylene Degreasers. CROWLEY, R. C., FORD, C. B., and STERN, A. C. (1945). *J. industr. Hyg.*, **27,** 140.

Perchlorethylene degreasers of the non-condensing type are now being used which rely on electricity as a source of heat and upon a pair of electrically operated liquid and vapour thermostats to control their operation. Tests made by Morse and Goldberg on 11 machines of this type yielded vapour concentrations in the breathing zone of the worker averaging 221 p.p.m., as compared with 135 p.p.m. for conventional non-ventilated condenser-equipped trichlorethylene degreasers, and 96 p.p.m. for ventilated degreasers of the same type. The accepted maximum limit for trichlorethylene and perchlorethylene is 200 p.p.m., and even this figure may be too high. In the present inquiry fifteen perchlorethylene degreasing tanks of the non-condensing type were investigated, and air tests made at eight of these tanks during operation showed perchlorethylene in the breathing zone of the operator averaging 180 p.p.m. when objects such as large and small castings were being put in the tanks, and 484 p.p.m. when they were being removed. It is therefore recommended that for safe operation this type of tank be used only when (*a*) equipped with condenser and local exhaust ventilation, and (*b*) operated according to all other generally accepted standards for safe degreaser operations.

Balances of Fluorine ingested from Various Sources in Food and Water by Five Young Men. MCCLURE, F. J., MITCHELL, H. H., HAMILTON, T. S., and KINSER, C. A. (1945). *J. industr. Hyg.*, **27,** 159.

It is well known that ingestion of fluoride compounds in abnormal quantities generally results in the extensive storage of fluorine in skeletal tissues, and it is stated that endemic dental fluorosis (mottled enamel) may arise if the domestic water supply contains more than 1·0–1·5 p.p.m. of fluorine. The present investigation was made on five young men who spent eight hours each day of the five-day test periods in an experimental chamber which was either maintained at the 'comfortable' temperature of 84° F. and of 50 per cent. relative humidity, or at the 'hot-moist' temperature of 100° F. and 68 per cent. humidity. The daily food ration provided contained 0·5–0·9 mg. fluorine, together with additional supplements in food and water providing 3·2–5·0 mg. The fluorine excreted in the sweat and body washings, and in the urine and faeces, was determined, and it was found that the two contrasted sets of environmental conditions had a marked influence on excretion. Of the fluorine eliminated by the kidneys and skin, 77 per cent. of that absorbed in the 'comfortable' periods appeared in the urine and 24 per cent. in the perspiration, whilst in the 'hot-moist' periods the averages were 49 per cent. in the urine and 44 per cent. in the perspiration. The (largely visible) sweat collected in the hot-moist periods contained 0·31 p.p.m. of fluorine, while the insensible perspiration of the comfortable periods averaged 1·2 p.p.m. The absorption of fluorine from bone meal, cryolite and calcium fluoride (in food) was appreciably less than that from sodium fluoride in food or water, so it is evidently dependent on the solubility of the fluoride. The investigators found that the elimination of absorbed fluoride via the urine and perspiration is practically complete when the quantities absorbed do not exceed 4·0–5·0 mg. daily, so they consider that any drinking water which does not contribute more than 3·0–4·0 mg. daily is not likely to create endemic cumulative toxic fluorosis.

Fluoride Domestic Waters: Relation to Fracture, Height and Weight. MCCLURE, F. J. (1944). *U.S. Publ. Hlth. Rep.*, **59,** 1543.

An investigation into the relationship of fluorine ingestion and height, weight and bone fracture experience of selected groups of 1458 high school boys and 2529 young adult men entering U.S. armed forces was carried out. The fluorine content of the domestic waters varied from 0 in Washington, D.C., and New Hampshire to 5·0 p.p.m. in Texas. Texas men averaged 69·6 in. in height and 149·0 lb. in weight, whereas Washington, D.C., men averaged 69·3 in. and 151·2 lb., and those from New Hampshire 67·3 in. and 149·6 lb. These height-weight figures show no relation to fluorine exposure. Among high school boys the number of fractures per 100 boys varied from 21·3 to 32·4. At induction centres for the armed forces bone fracture experience was from 25·0 to 30·0 fractures per 100 men averaging about 18–25 years of age. While these data on bone fracture experience do not permit final conclusions, they do suggest that no serious impairment in skeletal performance, as might be manifest in the number of broken bones, seems related to exposure to fluoride domestic waters.

Fluoride Domestic Waters and Urinary Fluorine Excretion. MCCLURE, F. J., and KINSER, C. A. (1944). *U.S. Publ. Hlth. Rep.*, **59,** 1575.

Pooled specimens of urine from men examined at U.S. army induction centres were examined. Those obtained from men coming from fluoride areas were compared with those from men from non-fluoride areas. In Washington, D.C., and New Hampshire, the fluoride content in domestic waters was 0, in Indianapolis 0·2 p.p.m. and in Hendricks County, Ind., 0·6–1·8 p.p.m., whereas in Amarillo, Texas, it was 4·3–5·1 p.p.m., in

Lubbock, Texas, 3·8 p.p.m. and in Joliet, Ill., 1·3 p.p.m. When domestic waters are free of fluorine, the fluorine present in urine averages 0·3–0·5 p.p.m. Fluorine in the urine specimens is strikingly proportional to the fluorine content of the drinking water. In Hendricks County the fluorine present in the urine is 1·0 p.p.m., in Amarillo 4·0 p.p.m., in Lubbock 4·2 p.p.m. and Joliet 1·0 p.p.m. The close correlation between fluorine in drinking water and fluorine content of urine suggests that the presumed hazard of cumulative toxic bone fluorosis surrounding certain water-borne sources of fluorine in the United States is greatly reduced by this relationship. An efficient urinary elimination of fluorine appears to be characteristic of individuals residing in certain fluoride areas of the United States. The metabolism of fluorine under these conditions seems to be a normal function of the human body and seems characterized by a condition approaching metabolic equilibrium.

Toxicity and Potential Dangers of Aerosols, Mists and Dusting Powders containing DDT. NEAL, P. A., VON OETTINGEN, W. F., SMITH, W. W., MALMO, R. B., DUNN, R. C., MORAN, H. E., SWEENEY, T. R., ARMSTRONG, D. W., and WHITE, W. C. (1944). *U.S. Publ. Hlth. Rep.*, Supp. No. 177.

When dogs, rats and guinea-pigs were exposed to 54·4, 12·44 and 6·22 mg. of DDT per litre of air in a static atmosphere for 45 minutes, no toxic signs or symptoms resulted. In the aerosol containing not more than 6 per cent. sesame oil mice tolerate concentrations of 6·22 mg. of DDT per litre of air. If there are higher concentrations of either, after a few hours increased excitability, nervousness, tremors and clonic convulsions develop, and later death. The DDT contaminates the fur and enters the body through the animal licking the fur and by absorption through the skin. Daily exposure of monkeys and mice for 45 minutes over a period of 5 weeks to an intermittent concentration of 0·183 mg. DDT per litre of air caused no toxic effects. When the exposure was repeated three times daily for 4 weeks, monkeys showed no toxic effects, whereas mice showed definite signs of poisoning. Exposure of two human subjects to 0·035 mg. of DDT per litre of air for 1 hour daily for 6 days, produced no toxic effects, nor did three times this concentration. DDT sticks firmly to any surface and therefore it was not possible to build up a higher concentration. Daily exposure for 3 hours over a period of 4 weeks to concentrations of 12·48 mg. of DDT dust per cubic metre of air caused no toxic effects in dogs. Daily insufflation of 100 mg. per kg. of pure DDT caused primary injury to the liver and kidneys and subsequent nervous manifestations resulting in tremors in one out of three dogs. Daily oral administration of 100 mg. per kg. of pure DDT over a period of 7 weeks caused no toxic symptoms in dogs beyond a moderate increase in the daily volume of urine. Exposure of rabbits to 1 per cent. DDT heavy mist for 48 minutes daily resulted in irritation of the eyes and upper respiratory tract caused by the solvent. Pathological findings in animals exposed to high concentrations of DDT included fatty degeneration of liver and kidneys in eight mice, three showed a slight degree of centro-lobar changes in the liver. Most of the animals showed a moderate degree of haemosiderosis and the anterior motor neurones showed evidence of degeneration. The liver of one dog out of four which had symptoms of DDT poisoning showed a healing subacute yellow atrophy of the liver. One chemical and one spectrophotometric method for the determination of DDT are described. The x-ray diffraction pattern of DDT is also described.

Acute and Subacute Toxicity of DDT (2, 2-bis (p-chlorophenyl)-1, 1, 1-trichloroethane) to Laboratory Animals. WOODWARD, G., NELSON, A. A., and CALVERY, H. O. (1944). *J. Pharm. exper. Therap.*, **82,** 152.

DDT is acutely toxic by mouth to rats, mice, guinea-pigs, rabbits and chicks in doses ranging from 150 to 750 mg./kg. The symptoms produced in those animals which did not die consisted of anorexia, nervousness, sensitivity to stimuli and fine tremors. When given in small amounts in the diet (0·05 per cent. for rats and mice, 0·1 per cent. for guinea-pigs and 0·05 per cent. for growing chicks) for periods up to 20 weeks, signs of subacute toxicity arose. They consisted of hyperirritability, fine tremors, depression, inability to carry out voluntary movements and decreased food consumption. There is a wide variation in individual susceptibility and this makes estimation of a safely tolerated dose extremely difficult.

The Percutaneous Absorption of DDT (2, 2-bis (p-chlorophenyl) 1, 1, 1-trichloroethane) in Laboratory Animals. DRAIZE, J. H., NELSON, A. A., and CALVERY, H. O. (1944). *J. Pharm. exper. Therap.*, **82,** 159.

Powders containing DDT 5 per cent. and talc 95 per cent. applied to the skin produced no toxic symptoms. Thirty per cent. and 25 per cent. solutions of DDT in dimethyl and dibutyl phthalates are absorbed through the skin and produced symptoms of anorexia, loss of weight, hyperexcitability, and nervous tremors at a dosage level of 9·4 c.c./Kgm. Higher dosages produced clonic convulsions and in some cases produced death after 30 days. Animals with the most severe symptoms showed a moderate polymorphonuclear leucocytosis. There were wide individual variations in susceptibility.

A Case of DDT Poisoning in Man. WIGGLESWORTH, V. B. (1945). *Brit. med. J.*, **1,** 517.

A man of 30 allowed small quantities of an acetone solution of DDT to evaporate on the back of his hand. He developed a feeling of heaviness and aching in all the limbs, with weakness in the legs and spasms of extreme nervous tension. After 3 weeks sleep became impossible, and after another 6 days involuntary muscular tremors occurred over the whole body. His recovery was slow and was not complete after one year.

Isopropyl Alcohol: Rate of Disappearance from the Blood Stream of Dogs after Intravenous and Oral Administration. LEHMAN, A. J., SCHWERMA, H., and RICKARDS, E. (1944). *J. Pharm. exper. Therap.*, **82,** 196.

The concentration of isopropyl alcohol was determined in blood and body fluids by distilling off the alcohol under reduced pressure into a solution of potassium dichromate in strong sulphuric acid. The reduced dichromate was estimated iodometrically. The disappearance of isopropyl alcohol from the blood stream of dogs after a single intravenous administration does not proceed at a constant rate and is not directly proportional to the blood concentration. It is dependent upon the functional efficiency of all excretory organs. High concentrations were found in saliva, urine and stomach contents. It is rapidly absorbed from the gastro-intestinal tract and is widely distributed throughout the body in less than two hours. The narcotic action is twice that of ethyl alcohol.

The Systemic Nervous Affinity of Tricresyl Phosphate (Jamaica Ginger Palsy). ARING, C. D. (1942). *Brain*, **65,** 34.

Tri-*ortho*-cresyl phosphate poisoning is described clinically and pathologically. More than one hundred patients have been examined in the late stage of the palsy; and material for histological examination was obtained from 36 patients. The pathology may be divided into neural and extraneural. The small arteries, particularly the capillaries and pre-capillaries of muscle had extremely narrow lumina because of thickening of all of the coats of the vessels. The intima was involved to a greater degree than the media and adventitia. The larger arteries were relatively normal. The picture is similar to thrombo-angeitis obliterans and two patients had to have an amputation of the thigh for gangrene of

the feet. Degeneration in muscle could be related to the affected vessels with narrowed lumina. Degeneration of nerves was found in all cases. Axones were of irregular width and decreased in number, and there was severe patchy loss of myelin in the nerves. There was heavy overgrowth of connective tissue in nerve trunks. In the spinal cord the lepto-meninges were always thickened. Degeneration in white matter varied. There was always involvement of the lateral pyramidal tracts with degeneration most severe in the lower segments of the spinal cord. There was moderate involvement of Goll's column, most obvious in the upper segments. Anterior horn cells were numerically reduced. No abnormality was seen in brain stem or brain. Histological studies of one patient who recovered showed changes limited to the spinal cord. Slight demyelination of Goll's column, and a rim of comparatively light staining of myelin sheaths about the circumference of the cord, and a striking loss of anterior horn cells were the most marked changes. All of the small arteries of the viscera were affected by endothelial proliferation. One patient died of bulbar palsy. The rarity of this manifestation may be related directly to the limited amount of the exogenous toxin ingested. The single patient was unique in having taken 15 times the amount of the poison ingested by the ordinary patient. She died after 50 days, unable to talk or swallow. From the clinical standpoint poisoning from tri-*ortho*-cresyl phosphate resembles amyotrophic lateral sclerosis.

Subglottic Stenosis after Exposure to a High Concentration of Screening Smoke (2 mc. Chloride). LUMSDEN, R. B., and WEIR, C. D. (1945). *Brit. med. J.*, **1**, 554.

Seven casualties, three fatal, occurred from exposure to screening smoke following the explosion of smoke generators. One man developed cough with profuse frothy sputum, slight stridor and vomiting. Rhonchi were present. His temperature was 101·8°, pulse 135, and respirations 35. He later developed retrosternal pain and frothy salmon-pink sputum. Two days later blisters containing straw-coloured fluid developed on the ears and hands. The bronchitis resolved in five days, but three weeks later he developed stridor, followed by right basal pneumonia. In the middle of the next month he had marked oedema of arytenoids and ventricular folds with stricture of the glottic aperture. He was treated by tracheotomy and dilatation of the stricture by means of Hegar's dilators.

Nitrobenzene Poisoning from Furniture Cream. CHAPMAN, W. M., and FOX, C. G. (1945). *Brit. med. J.*, **1**, 557.

A man aged 45 swallowed a quantity of furniture cream by mistake at 8.30 a.m. He vomited half an hour later. At 3 p.m. he was drowsy and had marked cyanosis of the lips and conjunctivae. Later 3 pints of blood were withdrawn and he was given a transfusion of 3 pints of Group O blood. The nitrobenzene content of the furniture cream was 5 per cent.

Benzene Poisoning. OLDFELT, C. O. (1944). *Acta med. scand.*, **119**, 380.

This article gives an account of blood changes found among 180 males engaged in the rotogravure printing industry. Before 1940 a mixture of toluene, xylene and benzene had been used as a solvent in the industry; during the years 1940–43, the use of benzene had to be increased in Sweden, owing to the shortage of xylene and toluene. After 1943 xylene was made available in larger quantities. The author, however, does not know when and to what extent benzene was substituted for xylene and toluene in the different industries. A diagnosis of chronic benzene poisoning was made when the blood picture showed definite leucopenia or thrombopenia. Sometimes anaemia was the only abnormal feature. Thirty-eight persons showed chronic benzene poisoning, while of the remaining cases there were symptoms of acute poisoning in the past history in 61 instances, while in 81 there had been neither symptoms of acute intoxication nor evidence of blood changes. In this series anaemia was present in 25 of the cases, leucopenia in 35 and thrombopenia in 11. No counts showed lymphocyte values over 60 per cent. Eosinophilia was present in 3 cases. In 10 cases red cells showing punctate basophilia were observed in the peripheral blood. Sternal puncture was done in 6 cases. In each instance the specimen was rich in cells, erythropoiesis was very active and leucopoiesis was without any striking change. One patient became feverish; he had a gingivitis and died of agranulocytosis.

Discussion on the Influence of Nutritional Factors in Liver Disease. HIMSWORTH, H. P. (1945). *Proc. roy. Soc. Med.*, **38**, 101.

The suggested mechanism of massive necrosis seen in some TNT workers is best explained by consideration of the results of feeding rats with selenium. Rats fed on grain grown on soil rich in selenium develop an hepatic necrosis and nodular hyperplasia apparently identical with that seen in rats in a low protein diet. Thus selenium necrosis is prevented by the addition of casein or methionine to the diet. In the protein of plants grown on soil rich in selenium, it is found that this element has replaced the sulphur in the methionine and apparently such selenium-amino-acids are inutilizable. TNT combines with amino-acids. It is suggested that this combination is inutilizable. These are examples of what may be called conditioned amino-acid deficiencies.

Liver Injury in Dogs exposed to Trichlorethylene. SEIFTER, JOSEPH (1944). *J. industr. Hyg.*, **26**, 250.

Dogs were subjected to repeated and prolonged inhalations of trichloroethylene vapour of known concentrations, simulating factory exposure. The exposures were made in a galvanized-iron cylinder fitted with a plate-glass top, and the air-trichloroethylene was drawn through it at the rate of 350 litres per minute. The 17 dogs used were exposed to air containing from 150 to 750 parts per million of the solvent for 5 or 6 days a week over a period of 2–8 weeks. Liver function was measured every 10 days by the bromsulphalein test, and it showed a progressive impairment. The amounts of bromsulphalein retained in the blood 30 and 60 minutes after it was injected were larger than in normal dogs, and the retention was more pronounced when the exposure was more intense. The ability of the liver to conjugate chloral was also impaired, the chloral hydrate being administered by stomach tube. Microscopic sections of the liver taken from the dogs that died or were sacrificed during the period of intoxication showed depletion of glycogen and hydropic parenchymatous degeneration. Other signs of intoxication were anaemia, vomiting and diarrhoea.

Study of a Fatal Case of Chlorohydrin Poisoning. DIERKER, HUGH, and BROWN, PAUL G. (1944). *J. industr. Hyg.*, **26**, 277.

A healthy man, aged 61, cleaned some trays with anhydrous ethylene chlorohydrin, and though he wore impervious rubber garments he suffered so much from nausea and vertigo that after about two hours' exposure he had to cease work. A few hours later he became cyanotic, with laboured breathing, and 9 hours after the exposure he died. Tests made at the breathing level of the man, when cleaning, showed that the air contained about 1 mgm. per litre of the solvent. On exposing six white mice to an atmosphere containing 1·2 mg. per litre of the solvent for 2 hours they all became ill, and one of them died. The others recovered and seemed normal 24 hours later. Evidently ethylene chlorohydrin is a highly toxic substance.

Carbon Tetrachloride Poisoning; Report of Cases. KONWALER, B. E., and NOYES, C. B. (1944). *Calif. West. Med.*, **61**, 16.

An account of three cases of poisoning, one of which was fatal, is given. Three men who had drunk heavily over the week-end worked on Monday morning in a room measuring $10 \times 11 \times 50$ feet from 8.30 to 11.30 a.m.

Three men who had not had previous alcohol also worked in the room; these men developed no symptoms. They cleaned machinery with rags soaked in carbon tetrachloride and used 1·4 litres in the three hours. The smell was well marked. One man felt sick at 10 a.m. and he died; the second did not feel any symptoms until 6.30 p.m. that evening. The third who frequently left the chamber showed no symptoms until the following morning.

Effect of 3, 4-Benzpyrene on Regenerating Epidermis of Mice. SILBERBERG, M. and SILBERBERG, R. (1944). *Arch. Pathol.*, **38**, 215.

Benzene hastened wound healing by intensifying epithelial proliferation in mice. The mitotic index was increased and the migration of cells into the defect was hastened as compared with control animals. Benzpyrene applied to regenerating epithelium for 5 days hastened healing to the same degree as benzene. The mitotic index rose to ten times normal compared with six times for benzene. If benzpyrene was applied for 4 weeks it was still active when the stimulus to regeneration had ceased. In spite of increased epithelial proliferation, the complete closure of wounds in benzpyrene-treated animals lagged behind that observed in the benzene-treated mice, although it was hastened as compared with wound healing in control animals.

Influence of Bromobenzene on the Induction of Skin Tumours by 3, 4-Benzpyrene. CRABTREE, H. G. (1944). *Cancer Research*, **4**, 688.

Batches of 30 mice were treated with benzpyrene and a mixture of benzpyrene and bromobenzene; and it was shown by local applications of bromobenzene to the skin four times weekly that the carcinogenic action of benzpyrene can be inhibited and sometimes prevented. That the effect of bromobenzene is local was shown by applying the two substances to different parts of the body when no inhibitory effect took place. The effect is probably due to intermittent interference with sulphur metabolism. Bromobenzene is detoxicated by mercapturate formation in the skin before being excreted in the urine. Glutathione and ascorbic acid levels in the skin are quickly lowered by bromobenzene but recover in a few hours to normal levels.

Effect of Sodium Citrate on Uranium Poisoning in Dogs. GUSTAFSON, G. E., KOLETSKY, S., and FREE, A. H. (1944). *Arch. intern. Med.*, **74**, 416.

Ten pairs of bitches were studied. One of each pair received intravenously 5 mg. of uranyl nitrate per kilogram of body weight, and the other uranyl nitrate intravenously and sodium citrate, either orally or intravenously. All the dogs receiving only uranyl nitrate had severe tubular necrosis of the kidneys and died in renal failure. The blood urea rose to 360 mg. per 100 c.c. on the tenth day, the blood creatinine rose to 25·8 mg. per 100 c.c., while the serum chlorides fell to 61·2 milliequivalents per litre; anuria developed on the fifth day. The oral administration of sodium citrate afforded considerable protection against the uranyl nitrate. In these dogs, at the end of 10 days, the blood urea was 9·4 mg. per c.c., blood creatinine 1·2 mg. per 100 c.c., serum chloride 103·2 milliequivalents per litre and urea clearance 42·4. In the urine of 7 dogs given uranyl nitrate only the amount of uranium excreted during the first 24 hours was 15 per cent. of the amount given as compared with 58 per cent. in the citrate treated animals. A brown precipitate of uranium was found in necrotic debris obstructing the lumens of the descending segments of the proximal convoluted tubules of the kidneys of the dogs having uranyl nitrate only, while little was found in the kidneys of the citrate treated animals. The protection of the kidneys from uranium poisoning by sodium citrate does not depend on correction of acidosis, since the latter follows rather than precedes severe renal damage. It is probably explained by the fact that the citrate facilitates excretion of the poison, thereby reducing its injurious effect. A factor possibly related to increased excretion of uranium concerns the *p*H at which uranium salts cause precipitation of serum proteins. The approximate range lies between *p*H 7 and 7.

Mule Spinners' Cancer and Automatic Wiping-down Motion. Interim Report of Joint Advisory Committee of the Cotton Industry (1945). H.M.S.O. London. Pp. 8. 2*d*.

The most certain method of preventing the onset of spinners' cancer is to use only such oils for lubrication as are, as far as is known, non-cancer-producing. Only animal and vegetable oils come within this category, but these are not available at present. The price of such oils should not be a difficulty, for, if non-splash methods are adopted only small amounts need be used in the industry. Mineral oils having certain physical characteristics are found to be least carcinogenic. Such oils, conforming to a certain specification, are now available and are being used by many firms. The specification is given as an appendix to the report, and the authors strongly recommend that only oils conforming to this should be used for the lubrication of spinning mules, until such time as non-carcinogenic oils are again available. That there should be periodic medical examination of all persons engaged in mule spinning is another important recommendation: such examinations to take place every 6 months under conditions of privacy for the worker and the doctor; records are to be strictly confidential but H.M. Medical Inspector of Factories is to have access to them.

Medical Examination of Employees in Hotels, Restaurants, and Other Food Places. WILSON, ARTHUR (1945). *Canad. publ. Hlth. J.*, **36**, 111.

An Order in Council, which became effective from July 1, 1944, in Saskatchewan, makes it 'compulsory for the employee of every hotel, restaurant, cafe, lunch counter, ice cream parlour and refreshment room where food or drink is sold to furnish annually to the owner or manager a certificate stating that he or she is not suffering from a communicable disease in a communicable state, and that he or she shows evidence of recent immunity to smallpox, diphtheria, and scarlet fever.' The Order applied to roughly 600 persons in Saskatoon, a town of 42,000.

'The first step for each employee was a visit to the venereal clinic at the City Hospital for the presumptive Kahn test and smear. If the Kahn test was positive, it was confirmed by a Wassermann. Smears were taken from the vagina, cervix and urethra of all females, but no prostatic smears were taken from the males.

'Following this, the employee received the vaccination for smallpox and the Schick, Dick and tuberculin tests. These were done in the one day. He or she was required to report the next day to have the Dick test read and the reaction to the Schick control noted; on the second day, for reading of the tuberculin test; and in about one week for the reading of the Schick test. The reaction to the smallpox vaccination was noted at each visit. Persons having positive Schick or Dick tests received the inoculations according to the usual practice. Those with positive tuberculin tests had appointments made at the sanatorium for an x-ray.'

Up to date (March 1945) nearly 500 certificates had been issued and a brief analysis of the results of the first 338 examinations is given. No positive TB cases were found; four men and five women were found to have positive Wassermann and Kahn reactions and five women were found to have positive smears for gonorrhoea.

The Saskatchewan Government has also passed similar legislation for medical examination of all operators in beauty parlours and barber shops, to be effective as from July 1, 1944.

PROCEEDINGS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

FORTIETH MEETING

The annual general meeting was held on October 19, 1945, at the London School of Hygiene and Tropical Medicine, Dr. J. C. Bridge in the chair. Following the private business, Dr. Bridge delivered his valedictory address as retiring chairman (reported below), and Mr. H. E. Griffiths (London) showed a film describing the treatment and rehabilitation of workers in the Royal Ordnance Factories of the Ministry of Supply during the late war. In the evening a dinner of the Association at the Trocadero Restaurant was held at which the chief guest was the Rt. Hon. Aneurin Bevan, M.P., Minister of Health.

Office Bearers, 1945–46

Dr. W. Blood (London) was elected Chairman and remains in office until October 1947.

Dr. P. Pringle (London) was elected Honorary Secretary.

Dr. Patricia Shaw (London) was elected Honorary Treasurer.

Drs. A. J. Amor (London), J. G. Billington (Birmingham), J. C. Bridge (London), T. A. Lloyd Davies (Nottingham), C. L. Potts (Redditch), and R. S. F. Schilling (London), were elected as the Executive Committee, together with the officers of the Association.

Messrs. Ogden, Hibberd, Bull and Langton, Chartered Accountants, were re-appointed as Auditors to the Association.

Standing Committees for 1945–46

Foreign Relations.—Drs. Elizabeth Bunbury (London), W. E. Chiesman (London), A. Forgie (Dagenham), R. E. Lane (Manchester), T. G. Maitland (Liverpool), D. C. Norris (London), P. Pringle and R. S. F. Schilling.

Industrial Nursing.—Drs. J. G. Billington, P. Pringle, Patricia Shaw, and Catherine Swanston (London).

Representatives on Outside Bodies

Birmingham Accident Hospital and Rehabilitation Centre.—Dr. W. J. Lloyd (Birmingham) was appointed a member of the Board of Management for three years as from February, 1945.

British Council for Rehabilitation.—Drs. N. T. Glynn (Birmingham) and C. L. Potts.

B.M.A. Industrial Medical Service Sub-committee.—Drs. W. Blood, J. C. Bridge, G. E. Graves-Peirce (London), and M. W. Goldblatt (Manchester).

British Standards Institute: Sub-committee on Welders' Goggles.—Dr. G. F. Keatinge (Derby).

Central Council for Health Education.—Dr. J. A. Mekelburg (London).

Central Council for Physical Education.—Dr. J. B. W. Rowe (London).

Empire Rheumatism Council.—Dr. T. A. Lloyd Davies.

Institute of Ophthalmology: Industrial Sub-committee.—Dr. H. E. Chard (Dagenham).

Minister of Labour's Industrial Health Advisory Committee.—Dr. M. W. Goldblatt.

National Advisory Committee for War-time Rest Breaks.—Dr. Patricia Shaw.

Research Board for Correlation of Medical Science and Physical Education.—Dr. W. E. Chiesman.

Education Committee's Report

The Chairman announced that the report of this Committee had now been published (see this JOURNAL, 1945, **2**, 158). The Association owed a great debt to the members of the Committee and their task was now discharged. The Hon. Secretary reported that there had been an extensive demand for copies from all over the country. The chairman of the Committee, Dr. W. E. Chiesman, stated that the report had been favourably received by the medical press, and by *The Times*. One of the recommendations was that the Royal Colleges should consider the institution of a Diploma in Industrial Health. In this connexion Dr. Bridge mentioned the fact that the Society of Apothecaries of London had now decided to institute a Diploma in Industrial Health,* but this decision was independent of the Committee's recommendations. Dr. Donald Stewart (Birmingham) mentioned the importance of that recommendation which states that training of nurses for industry should become an important part of the work of University Departments of Industrial Health; it was necessary that co-ordination of this work should occur and he stressed the need for the institution of a new central body to lay down standards of training and arrange for examination for the Certificate in Industrial Nursing. On this body should be representatives of the Association.

Finance

The Honorary Treasurer presented the financial statement for 1944–45, which was unanimously approved and adopted. The Association was in a sound financial position; on the other hand, 17 Ordinary Members and 18 Associate Members had not yet paid their subscriptions for the current year. In addition, 7 Ordinary Members were more than 18 months in arrears. Since many of these might be in H.M. Forces it had been difficult for him to decide on taking more serious steps to collect arrears under Rule 7 of the Constitution, but under this Rule, and by the unanimous approval of the meeting, the name of one member was removed from the list. It was agreed that the sum of 10 guineas be voted to the Association's Prize Fund for Industrial Medicine held by the London School of Hygiene, and that 25 guineas be voted for clerical assistance rendered during the year. It was also decided that the annual subscriptions for 1945–46 should remain at one guinea for Ordinary Members and half a guinea for Associate Members.

Committees of the Association

It was agreed, on the proposal by Dr. Bridge, seconded by Dr. Donald Stewart, that, in future, committees of the Association should be of two types: (*a*) Standing Committees instructed to deal with long-term programmes, and elected by Ordinary Members at a general meeting; (*b*) Sub-committees of the Executive Committee, instructed to deal with short-term or *ad hoc* programmes, and nominated by the Executive Committee. Both committees would report in the first instance to the Executive Committee, whose responsibility it would become to report thereafter, as it felt was necessary, to a general meeting of the Association. Discussion took place as to the correct placing of such committees as were already in being. The Committee on Industrial Nursing, and the Committee on Foreign Relations, were both approved as Standing Committees. The chairman reported that the Executive Committee had set up a sub-committee to study problems of industrial health as a whole and to advise on current trends—really a development sub-committee. Dr. W. A. Mailer (London) supported by Dr. J. Tarsh (Liverpool) felt that this should be a Standing Committee. Dr. M. W. Goldblatt, Prof. R. E. Lane and Dr. T. A. Lloyd Davies

* See *Brit. med. J.*, 1945, **2**, 480.

pointed out that this matter of development and the study of new trends was surely the responsibility of the Executive Committee itself and that the sub-committee would act only in an advisory capacity. It was finally decided that the committee in question should be a sub-committee of the Executive. As a corollary it was also agreed that it could not survive its parent body, the Executive Committee itself.

Royal Institute of British Architects

The Royal Institute of British Architects invited members to attend a meeting of the Institute to discuss 'The Construction of an Underground Factory.' Dr. W. E. Chiesman was nominated to attend on behalf of the Association and asked to report proceedings at a later date, in view of the importance of the subject in relation to the health of the worker.

Spens Committee

The Inter-departmental Committee on remuneration of medical practitioners, under the chairmanship of Sir Will Spens, had invited the Association, through the Ministry of Health, to give evidence on conditions of service and salaries of industrial medical officers. The general feeling of the meeting was these matters were not within the true functions of the Association, although members were reluctant not to be of assistance. It was agreed that the Honorary Secretary be instructed to send the list of members of the Association to the Committee, explaining the position but at the same time inviting the Committee to approach individual firms and individual members should they desire to do so.

Dinner

In accordance with the Constitution of the Association, and with a post-war policy which will be adhered to whenever possible, a dinner was held at the Trocadero Restaurant, Piccadilly Circus, after the meeting. The Rt. Hon. Aneurin Bevan, Mr. H. E. Griffiths, Sir Wilson Jameson and Brig. G. S. Parkinson were the guests of the evening. The newly elected Chairman of the Association, Dr. W. Blood, presided. Dr. A. J. Amor proposed the toast of the guests. He made special reference to the need for medical supervision in the mines of South Wales, and referred to the first-hand experience the Minister himself had had there in his youth. Mr. Bevan replied on behalf of the guests. In his view industrial medicine was a vital branch of medicine and there was an urgent need to provide medical care at the place of work, not only for large firms but for the thousands of small units in industry. Industrial health must be integrated with national health and in this respect the general practitioner must play an increasingly important part in any scheme that might be developed.

SOME THOUGHTS AFTER THIRTY YEARS IN INDUSTRY *

BY

J. C. BRIDGE

Having had something to do with industry during two great wars there is certainly one good thing that in my opinion has emerged from them, and that is the increasing regard which has been paid by the Government, employers and people in general, to the health of the workers. These men and women have done a task which has enabled us to look forward to the avoidance of another war which could well decimate our children and our children's children. We must not, however, forget that in the peaceful times even before the war of 1914–18 there were men practising industrial medicine not only in this country but abroad, for example Arlidge, Oliver, Rambousek and Glibert. By the practice of their profession and their writings these men had undoubtedly shown the important part that medicine must play in the well-being of industry. But I venture to suggest that so far as this country is concerned, the appointment of Whitelegge as Chief Inspector of Factories and the subsequent appointment of Legge as Medical Inspector of Factories were landmarks in the history of British industrial medicine, and did much to emphasize the essential role that medicine must play in industrial affairs.

So much has changed in industry since I was first closely associated with it thirty and more years ago that it is difficult to know where to begin. All that it seems possible for me to do is to pick out some of the outstanding features that occur to a single individual like myself.

One of the most striking things that I find changed is what I may perhaps call the general environment of the worker to which so much attention is now given. My friend Dr. Merewether in his Annual Report for 1943 says—'One can hardly fail to have noted in factories this enhanced consciousness of the importance of healthy living and interest in the means by which it can be achieved.' More and more have those concerned with industry appreciated the importance of environment, of matters such as tidiness in the shops, as adding to the comfort and well-being of the worker. Who, 30 years ago, would have thought of the safe passage marked by white lines in an engineering shop for example? In the environment of a worker I must include amenities such as washing accommodation, cloakrooms and the like. While the pail which could be filled with warm water in which the workman washed his hands is still extant, the more convenient forms of washing accommodation with hot and cold water laid on, with towels, soap and nail brushes are indeed more the rule now than the exception.

However much I may dislike the term 'welfare,' I feel that we owe not a little to the impetus given to industrialists for the provision of amenities by the activities of the society instituted and continued by my old friend Mr. Robert Hyde. There were few canteens, if they could even be described as canteens, 30 years ago. In my submission there has been no greater contribution to the well-being of the industrial worker than the establishment of canteens at his place of employment. This has been one of the main factors that enabled him to carry on throughout the hard and strenuous years of the late war. I must pay my tribute to my colleagues at the Ministry of Labour who took part in their establishment and to the Minister himself who was so far-seeing as to make them a requirement in factories concerned with the production of materials for war. I personally express the hope and belief that in the peace which we expect will eventually follow, industry will continue this service which is so important.

Some Toxic Hazards

Turning to the definite industrial hazards, industrial poisonings and diseases, and looking back to the days when lead, arsenic, phosphorus, mercury and anthrax were those diseases to which our special attention was directed, no little progress has been made in the recognition of other poisons and diseases which are contracted in industry consequent upon employment. Recognition, however, must precede prevention and the channels of the attack of the noxious substance must be

* Chairman's valedictory address delivered to the Association of Industrial Medical Officers at the London School of Hygiene on October 19, 1945.

established before we can approach prevention in an understanding manner. The outstanding example of the importance of recognition, both of the disease and of the channels of absorption of the causative agent, is exemplified by the work of Legge. By careful observation he not only showed that inhaled dust of a lead compound was the prime factor in the causation of lead poisoning, but also estimated that 2 mg. of lead in 10 cubic feet of air if inhaled daily would produce poisoning. As a result of this observation he advised preventive measures which reduced the number of cases of lead poisoning from over 1000 in 1900 to 72 when I left the Factory Department in 1942. Others, following Legge's example, have enumerated various air concentrations of toxic substances which will cause poisoning.

I must refer also to Legge's work on anthrax. Those of you who are interested in the subject must, I feel sure, have read his Milroy Lectures on this disease. He introduced into this country the sclavo serum, a romance of the prevention of the fatal consequences of an industrial disease; and indeed it was a true romance because he brought it back from Italy on his return from his honeymoon.

Thirty years ago there were those recognized poisonings and diseases to which I have previously referred but others have since been recognized. To-day some 43 diseases are on the compensation list. It is interesting, looking back, to find how the nomenclature of one condition fits in with a similar condition caused by quite a different substance. For example, in 1915 toxic jaundice was made a notifiable disease on account of its occurrence among workers using tetrachlorethane. Shortly afterwards cases of jaundice arose from exposure to TNT, and still later on this description fully covered poisoning by chlorinated naphthalene, to say nothing of the jaundice produced by the inhalation of arsine.

A grave hazard can be produced not only by the introduction of new chemicals into industry but also by the use of even well-known chemicals in a new process. In the rayon industry, for example, in which the cellulose is dissolved in carbon bisulphide, the use of carbon bisulphide gave rise to the possibility of serious illness and I have recollections, in the early days of this industry, of seeing several workmen suffering from chronic carbon bisulphide poisoning. More recent innovations into industry produced by research workers, for example the numerous organic solvents, the organic chemicals used in the dyestuffs industry, lead tetra ethyl, chlorinated naphthalenes, and the extensive use of luminous paints containing mesothorium, have all raised important health problems. In connexion with this last substance, I introduced the term ' toxic anaemia.' For the purposes of notification I was perplexed to find a term for the condition which was produced not only by the ray of radium but also by other substances; the anaemia was found to be of a dyshaemopoietic character. One has been concerned also to think of the potential hazards to workmen engaged in the preparation of the atomic bomb.

As to the mode of entry of toxic chemical substances we have some knowledge, but as to the reaction of these substances on the metabolism of the body once they get into the body we are in considerable ignorance, and it seems to me that until we can elucidate this problem we shall never really arrive at the true solution of the method of prevention. The recognition of carcinoma of the bladder arising in industry, following that of carcinoma of the skin, is historical, but with the introduction of new chemicals its significance has increased. I cannot leave the subject of industrial cancer without referring to the great work done on the subject by my colleague, Dr. Henry. It has always been my hope, which perhaps some day will be fulfilled, that the knowledge we obtain in industry of such diseases as cancer of the skin and of the various forms of anaemia, may lead eventually to the elucidation of the causes of these diseases in the general population, but so far my vision has not been realized.

When I joined the Factory Department 30 odd years ago, Professor Collis was then engaged on the study of silicosis as it occurred in this country. While I feel that every attention was given to his Milroy Lectures, it was unfortunate that they were delivered during the great war of 1914–18. Much progress has been made since then in the study of this disease, but while Collis did point to the possibilities of silicates causing lung fibrosis little was known until our attention was drawn to cases of lung disease occurring in asbestos workers in Scotland. A very full and careful inquiry made by Dr. Merewether established without question a definite pulmonary disease caused by the inhalation of the dust of asbestos. One of the great problems that has always faced us in connexion with silicosis has been the grinding of metals and the cleaning of castings and Dr. Middleton's work on this aspect of the problem is no doubt well known to you all. One of the most frequent causes of silicosis was the cleaning of castings by sandblasting, and I must confess that at first the alternative use of steel shot for sand appeared the solution. But it was not long before I realized I was mistaken, for reasons too detailed to consider now. I was more than glad to learn that the new code of regulations issued this year by the Ministry of Labour prohibits the use of sandblasting, but the problem of silicosis in the cleaning of castings is not yet solved—not even by the hydroblast—and the hazard in the fettling of such castings has yet to be prevented. I trust the Committee of the Factory Department which is now sitting will give their fullest attention to this matter in the near future.

The production of highly lethal chemicals, particularly for example lead tetra ethyl, organic mercury compounds and war gases, has necessitated the re-orientation of our ideas as to the proper method of dealing with hazards due to poisoning caused by fume and dust. My colleague in the Ministry of Labour, Mr. Wilkes, has recently addressed the Association on the necessity for complete enclosure of plant when dealing with highly toxic substances such as these. This method of dealing with processes of a high hazard should be developed and I feel sure that chemists and engineers concerned with industry are having and, will in the future, have regard to the elimination of all risk by these means.

Research

It seems right that in briefly reviewing the work of the past few years in industry one should mention the subject of research. It has always been my hope that the study of an industrial disease, similar in character to that occurring in the general population, should lead to some better knowledge of the disease and its prevention in the population as a whole. Research in industry must not only be a study of the disease and the mode of attack of the causative agent, but also the measures necessary for its prevention. For this research the medical officer, the chemist, the physicist and the engineer must form a co-operative team to work out the solution. While fully appreciating the work that has been done by the Industrial Health Research Board, the scope of this body must necessarily be limited. It is true that the activities of the Board have recently been extended by the setting up of the Department for research in industrial medicine at the London Hospital under Dr. Donald Hunter. In 1935 the Home Office asked the Medical Research Council to set up a committee for the investigation of industrial pulmonary diseases, but just at that time there arose the problem of a disease amongst coalminers atypical of silicosis and the need for compensation overshadowed other inquiries into pulmonary diseases occurring in industry which might then have well been made. We know little yet of the effect of so-called inert dusts on the lungs although considerable field work has been done by McLaughlin, Doig and others.

In 1936 the problem of the numerous organic solvents coming into use in industry and their effect on health was one which called for further investigation, and the Medical Research Council, at the request of the Home

Office, set up a committee known as the Committee on the Toxicity of Industrial Solvents under the chairmanship of Sir Joseph Barcroft. The committee's work was circumscribed and its progress was limited during the year or two that elapsed before the outbreak of this war. It was impressed upon me that no committee of this character could function effectively without a research department, so I had in mind the setting up by the Government of a research department which should be intimately associated with the Factory Department and to which industrialists and others concerned could bring their problems. There was, and there still is, a research department at Porton associated with chemical warfare which long before the war I visualized could be turned over to the solution of peaceful problems and the prevention of industrial ill-health. I still hold the opinion that the establishment of a central research department, controlled by the Government, is essential, but while saying this I do not for one minute suggest that industry itself should not continue to carry out research in its own spheres.

Compensation

While no doubt the new social insurance legislation dealing with industrial accidents will remove many of the disadvantages of the present system, I am not very happy about it all. It may be that I have a bee in my bonnet, but I am afraid that bee has been buzzing for a very long time. I take it that payment of compensation for an injury is not really compensation but secures for the injured workman relief of anxiety as to the economic position of himself, his wife and his family. We are, as medical practitioners, fully appreciative of the necessity for relieving anxiety not only in cases of injury but also of illness. I am therefore equally concerned with the ill workman who is suffering say from pneumonia or typhoid fever, and with his wife and family; he contracts these diseases, without any suggestion of contributory negligence at his place of work. Compare his case with a young adult who has a simple fracture arising out of his employment. I fail to see why the workman disabled by disease should not be relieved of his anxiety to the same extent as the workman disabled by accident. With the various Medical Boards and Tribunals that are to be set up there should, so it seems to me, be no difficulty in debiting any benefits paid to the man to the appropriate fund. I believe too that this would get rid of a large measure of mistrust towards our profession when we have to say to the workman, for example, that through no fault of his own he has a disabling skin disease not in our view due to occupation; in such a case we are obliged to tell him that he will get less relief from his anxiety. If the workman is to be insured let him be insured against sickness and accident at the same rate. Who of us can say that a bronchitis is in all cases definitely *not* due to a man's occupation? I am afraid I cannot.

Medical Supervision

I am going to be so presumptuous as to say that I regard myself as the father of medical supervision in industry in this country. True, for over 100 years the State has exercised some supervision of industry by the appointment of Certifying Surgeons—after the passing of the Factories Act of 1937 designated Examining Surgeons—but their functions are very limited and are mainly concerned with the examination of young persons. In my younger days, as a matter of interest, young persons included youngsters of 12 years who were employed as half-timers. Examining Surgeons were, and still are, employed to carry out periodic medical examination of workers engaged in processes covered by certain regulations dealing with occupations giving rise to a health risk, but I think most of the work of the Examining Surgeon should be performed by the medical officer who is responsible for the medical supervision in the factory. During the war of 1914–18, and also during this last war, an increasing impetus was given to medical supervision. The Ministry of Munitions in the 1914–18 war and the Ministry of Supply in this last war organized and established a pretty complete system of medical service. But we must not forget that during the intervening period private enterprise established, on an increasing scale, a medical service for their industries and I am going to be so bold as to say that these services in most instances cared for the health of the worker in a manner not inferior to that exercised by the Ministries. Not one of us can wish to see medical service in industry diminished, and this Association has on more than one occasion expressed the view that a medical service should be extended not only to cover places that make things but to all branches of industry and occupation. The Association too has placed itself at the disposal of any Department wishing advice on the formation of a plan for an organized industrial medical service, that is a service which would fit in with any jigsaw puzzle of a comprehensive national health scheme. Speaking for myself I trust that in the end whatever scheme is devised the individuality of the industrial service will not be lost, yet it is vitally important that it be linked with the wider health service which is now being planned. Those who would divorce the industrial service or attach it to local authority control ought to revise their views. I care not whether the control be lay or medical.

The Association has recently published a report on the education of the industrial medical officer. This, I suggest, indicates the special knowledge that it is desirable a medical officer in industry should have. In this connexion you may have seen that a Diploma in Industrial Health has now been set up by the Society of Apothecaries; surely this is a landmark in the history of our subject. In the next year or so there will be an opportunity to establish a medical service in industry in Great Britain which I firmly believe can be made second to none, whether it be in the U.S.S.R. or the U.S.A., or anywhere else. It is up to you young men to see that the service is formulated to do what is best for the British workman.

Rehabilitation

I cannot finish without a word or two on rehabilitation. During the war of 1914–18 efforts were made in this direction but they were limited and many faded out. It was a grief to me and although I made some attempt to keep them going for the workers I got little support. But to-day everybody is talking about rehabilitation. However, I warn you not to imagine that the ball is going to roll on without an occasional kick and sometimes it may have to be a very hard one.

The Future of the Association

In conclusion I come to a domestic matter—the future of the Association. It was undoubtedly necessary to widen admission to the Association by including those members of our profession who were only engaged for a part of their time in the practice of medicine in industry. This was achieved by the admission of Associate Members. I am doubtful, after very careful thought, whether it is wise to continue this differentiation and whether it is not now time to consolidate the Association. I doubt whether the primary object of the Association, namely the scientific study of our subject, is best met by full meetings of the Association with its increasing numbers. This view has already been partly accepted by the formation of local groups. The question is whether it should not be extended. So I suggest that the time has now come for the formation of a small elected Council to carry out the routine work of the Association, and that the full Association meetings should be limited to an annual or bi-annual gathering. It is a matter which I feel is of no little importance and with this thought I leave you.

Finally, may I thank you all for the great consideration you have extended to me as your chairman. So far as my years allow me I shall always be at your service.

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